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HANDBOOK

OF

MEDICAL TREATMENT

EDITED BY

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WITH THE ACTIVE CO-OPERATION
OF
FIFTEEN ASSOCIATE EDITORS

IN TWO VOLUMES

VOLUME ONE

ILLUSTRATED

WITH AN INTRODUCTION
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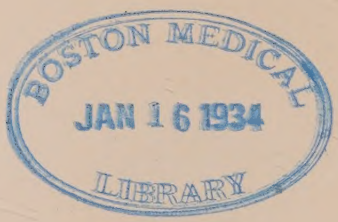


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PREFACE.

THIS book represents the correlated efforts of a representative group of Philadelphia medical teachers and hospital workers to present the principles of modern medical treatment in an authoritative manner. The endeavor has been made to prepare a practical working manual, unburdened by technicalities, free from useless tradition, and expressed with a directness and simplicity that reads fact for theory, and succinctly expresses the various writers' statements as the personal opinions and experiences of skilled consultants.

The editor has been most fortunate in having enlisted as his associates in the undertaking the interest of a staff whose personnel is an earnest of an *ex cathedra* presentation of the subjects treated, and who represent all that is final, sane, and progressive in the medical world today. Inasmuch as the intelligent treatment of any given disease must rest upon an appreciation of the underlying pathologic lesions and diagnostic data, the topics of clinical pathology and diagnosis receive commensurate emphasis. This makes for a clearer understanding of the therapeutic measures subsequently advised, which are considered upon the tangible basis of pathologic cause. Laboratory technic and complete clinical descriptions, not being germane to the plan of this work, are omitted save when their inclusion seems helpful to certain details of treatment.

The subjects dealt with are those of interest to the practitioner of internal medicine, and do not primarily concern the surgeon or the specialist. The classification of diseases is that generally conceded as most acceptable, but certain arbitrary exceptions to this general rule are made, notably in the acute infections, exanthemata, and tropical diseases, in order

thus to obtain the personal opinion of the author best qualified to direct the care of these disorders.

The editor is under obligations to the W. B. Saunders Company and to P. Blakiston & Sons Company for permission to use illustrations from his published works on Physical Diagnosis and Clinical Hematology; the latter firm has also allowed the reproduction of the figures from Schott's monograph on the Balneogymnastic Treatment of Cardiac Disease.

J. C. Da C., Jr.

INTRODUCTION.

One of the pleasures of an old teacher is to see his pupils growing in usefulness and service to our profession and to the community. Among the Associate Editors of Da Costa's book are several of my old pupils, including in this pleasant company the highly esteemed editor himself, in whose careers I take a genuine personal interest and a pedagogic pride. Those whom I have not had the pleasure and honor of teaching I have known or known of, and greet them as fellow-authors striving to elevate and instruct the younger and less experienced members of the profession, and to serve our Country, and also to serve our common Humanity. It is of interest to note that nearly all of the Associate Editors are already in the Medical Corps of our Army, Navy, or Public Health Service. The few not so enrolled have reasons valid to their consciences or to their countrymen.

I have seen all the proofs of the first volume, and so far as a surgeon can judge, it bids fair to be of very great practical value. The second volume, I hope, will be its chief rival.

W. W. Keen.

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Specific Infections

BY

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Specific Infections.

FOREWORD.

The section on the Specific Infections has been written with the hope and the expectation that it will prove helpful to the practitioner of medicine, and to this end the subject matter has been freed of technicalities as far as possible. Sufficient reference to clinical pathology and bacteriology has been made to give the reader a clear idea of the etiology of the various diseases considered, and to furnish a stable groundwork for the therapeutic measures advocated.

The treatment of the infectious diseases here described is based upon the writer's understanding of their clinical features, and in many instances the methods given represent his own plan of management in the circumstances met with. This course has been determined by his personal experience, supplemented by a rather wide consultation of the current literature, but originality is disclaimed, save that which naturally may be ascribed to one's practical experience in the ward and in the sick-room.

To the authors of numerous standard textbooks and monographs the writer owes much of the information herewith presented, and it has been his endeavor to give credit in the text for data from these sources.

TYPHOID FEVER.

In the management of typhoid fever one must have as fair an understanding as possible of the cause, the pathology, and the means of its transmission from one individual to another.

The specific cause of typhoid fever is a motile bacillus, the *Bacillus typhosus,* or Eberth's bacillus, a micro-organism which, after gaining lodgment in the body, attacks especially the lymphoid tissue, and almost without exception the lymphatic glands, solitary glands and Pyer's patches of the small

intestine, and also the lymph-glands in the cecum and in the first part of the large intestine.

There is a general engorgement and enlargement of the lymph-glands, especially of the mesenteric group, and of the spleen. There is a demonstrable bacteremia during the first two weeks of the disease, but the organisms are difficult to find in the blood-stream during the later weeks of the infection. After the first fortnight the typhoid subject becomes toxic, and the disease works its greatest destruction through its toxic effects, except when localization of the bacteria in such areas as the lymph-glands of the intestines, in the larynx, in the lungs, and in other situations excites local complications which may imperil the patient's life or prove fatal.

The typhoid bacilli are found during the disease, and often long after active symptoms have disappeared, in both the feces and the urine, and have been demonstrated in the saliva.

Knowledge of these latter facts is of the utmost value to everyone, and especially to the laity, because this knowledge gives absolute ability to prevent the disease.

The fact that typhoid fever is caused by a micro-organism which is thrown off in the feces, urine and saliva makes it possible to lay down rules for its absolute prevention. It is because this certain knowledge is not universally applied to every case that typhoid fever is so prevalent a disease, for destruction and disinfection of all of the excreta at their source would, in a short time, cause the absolute disappearance of the disease.

Methods of Prophylaxis. Every case of typhoid fever must be considered a source from which other cases may arise, and, without exception, the typhoid patient should be in the hands of one or more well-instructed nurses, or be treated in a hospital. The nurse should wear a wash gown and rubber gloves when handling the patient, the bed-clothing, or anything which comes in contact with the patient. When the gloves are not in use they should be kept immersed in some antiseptic solution. This care on the part of the nurse will prevent her from contracting the disease and communicating it to others.

The *feces* should at once be disinfected, as soon as passed. The bed-pan should contain either a 10 per cent. solution of

phenol or chlorinated lime, or the feces should be mixed with an equal bulk of boiling water. If an antiseptic is used it should be thoroughly mixed with the feces, and the mixture allowed to stand half an hour before being emptied into the hopper. This disinfection should be extremely carefully done, if an ordinary privy-well is connected with the house.

The same care should be taken in disinfecting the *urine,* for it is certain that this excretion contains active bacilli. An additional method of sterilizing the urine is to administer to the patient 5 grains (0.324 Gm.) of hexamethylenamin (urotropin) three times a day, during the whole course of the illness.

All discharges from the *mouth,* including the expectoration, should be collected on bits of cloth and immediately burned, or these bits of infected material may be put in a paper container, and this burned at the first opportunity.

All *bed-linen* is to be placed in a disinfecting solution and boiled before being put through the common laundry. All unused food should be burned or otherwise thoroughly disinfected. Any utensils used in the sick-room should be immersed in a disinfecting solution and boiled before being washed with the ordinary dishes, or handled by anyone except the nurse.

As stated above, if these rules were carried out in all cases, typhoid fever would soon become an unknown disease. Unfortunately, however, all cases are not observed in the first few days; some are not observed for a long time, and unquestionably many cases are never recognized definitely as typhoid fever. This fact, together with disregard of the well-known nursing rules, are the sources from which isolated cases of typhoid fever are constantly arising, and these cases, through the medium of drinking-water and contaminated food-supply, are multiplied, so that typhoid fever is an endemic disease. It is a well-known fact that well-regulated cities with filtered or otherwise purified water, a clean food-supply, and a good sewage-disposal plant are much less likely to be sources of infection from typhoid fever than the "healthy" country, where there is no sewage, and where dejecta, often not disinfected, are disposed of in open privies, close to the well which supplies the drinking-water, while the milk is supplied

from dairies that are either badly inspected or not inspected at all. Thus, at the end of every vacation season there is a more or less severe epidemic of typhoid fever in the cities to which have returned vacationists who have sojourned during the summer in localities infected in this manner.

Purification of the water-supply is perhaps the most important single factor in the prevention of epidemics of typhoid fever. Only one instance of this need be mentioned. Philadelphia was supplied with infected water from the Delaware and Schuylkill Rivers until 1906. Previous to the supply of filtered water to all citizens in Philadelphia the deaths from typhoid fever ranged between 666 and 1063 per annum. Since filtered water has been used the yearly number of deaths has steadily decreased, until in 1910, there were 270 deaths, and about 20 per cent. of these were traceable as to the origin of the case to communities outside of Philadelphia.

When one is not sure of the purity of the water one is forced to use, all water should be boiled. Another fertile method by which the disease is spread is *milk*. In some cities, a law more or less adequately carried out, demands that all milk dispensed by dealers should be pasteurized before being sold. Individuals who are uncertain of the cleanliness of their milk should always pasteurize their individual milk-supply.

Insects, especially flies, are fertile means of infection. They gain access to infected material, especially to the feces, their feet become covered with this material, they then crawl over food about to be eaten, and hence the germs are carried directly to the person eating the food.

Two lessons are to be learned from this: *First,* every possible precaution must be taken to disinfect feces, urine and sputum; and, *second,* the sick individual must be in a screened room, in order to prevent the flies getting to the original source of the infection, the patient.

Shellfish, and fresh, uncooked vegetables, such as lettuce and various greens, are possible sources of infection.

Last, but by no means least, is the typhoid carrier, by which is meant an individual who is constantly discharging from the intestinal tract living typhoid bacilli. The obvious lesson here is to see that both urine and feces are free from

typhoid bacilli before the patient is discharged. This, of course, can be done through the established laboratories common throughout the country.

Sir Almroth Wright, during the Boer War, attempted to immunize the British Army by means of the killed bodies of typhoid bacilli. This particular attempt at prophylactic vaccination failed in its effects, probably because of some error of technic, but later attempts have been most successful. In 1913, among the 90,000 American troops serving in all parts of the world, there was not a single death from typhoid fever in all this number. The figures of the present European War, and of the mobilized American troops on the Mexican border are not yet available, but it is reported that during the first months of the European War, before all the hastily assembled English forces were vaccinated, typhoid fever was a very common source of disability.

It has been proved repeatedly that concentrated amounts of living bacilli ingested by a person who has been vaccinated can produce typhoid fever in the inoculated subject. Hence, we must be careful in all the more familiar precautions of prophylaxis, and not neglect them because we have a potent preventive in vaccination.

Vaccination is most valuable, and should be employed even more largely than it is at present, but it should not be used to the exclusion of methods which destroy the disease at its source.

Prophylaxis, then, aside from preventive vaccination, consists, briefly, of the recognition of cases of typhoid fever in the very earliest stage, of careful nursing, of the disinfection of all excreta, of the use of uncontaminated water, milk, vegetables and shellfish, and of screening of the patient and excreta, so that flies and other insects may not carry typhoid germs to the food.

TREATMENT.

Complete rest, careful nursing, sufficient and well-selected food, control of the temperature, and early recognition and treatment of complications, with the possible use of typhoid vaccines, are the main points upon which one can rely to

carry the patient through this most treacherous and trying disease.

Rest. This means absolute quiet in bed, the patient not being allowed to rise even for the purpose of evacuating the bowels or for urinating. It is a well-known fact that typhoid fever begins in various ways, gradually or suddenly, and often with symptoms suggestive of disease of special organs in which it is impossible to make an early differential diagnosis. If all such cases were treated so far as rest is concerned as though they were typhoid fever, one would less frequently be confronted later with a well-developed case of typhoid when "enteritis" or "catarrhal fever" were thought to be present.

It goes without saying that an unremitting search for the cause of the symptoms should be made. This search should include repeated examinations of the blood, urine and feces. A bed-pan should be used for feces and urine. By thus conserving the strength of the patient, complications may be avoided.

This rest includes, of course, an abundance of sleep. Patients with typhoid fever should not be awakened for food, drink or medicine, unless they are so drowsy from the toxemia that they are stuporous and fall to sleep again immediately.

Efficient Nursing. There is no one element in the treatment of typhoid fever which is of as great value as the services of an efficient nurse. A well-trained nurse, or, much better, two nurses, should be in attendance on every case of this infection. If this, for financial reasons, is not practicable in the home, then the patient is much better off in a well-regulated hospital, where such services are at everyone's call. I have used the term "well-trained" in connection with the nurse. I do this because it must be remembered that, while the great majority of hospitals give such a training to their nurses that the efficiency of their services can be relied upon, this is not invariably the fact, and, therefore, because the physician must rely entirely upon a nurse to carry out his orders, and to watch for complications which may appear suddenly in the mildest cases, it becomes the physician's duty to see that the nurse comes from an institution in which the

training is adequate, and also to discover, if possible, the character of the individual nurse before she is employed.

It is the duty of the nurse to administer the food, to bathe the patient, to administer the drugs according to the directions and orders of the doctor, and especially is it her duty to report to the physician at once any untoward circumstance. She should be able to recognize symptoms indicative of perforation, hemorrhage, sudden cardiac weakness, and similar complications, and should at once report them to the physician, and not presume to administer treatment herself.

Food. The food of typhoid patients should be of sufficient quantity to nourish the patient, and should be of such a character that it can be properly digested and will not irritate the intestinal tract.

Coleman and DuBois have shown that the metabolism of patients ill with typhoid is not diminished, and their experience, together with that of many other clinicians, has proved that food which contains a daily intake of from 2500 to 3000 calories serves so to maintain the strength of the patient that he loses minimum weight, that his convalescence is much hastened, and that complications are less severe.

A milk diet, which perhaps is the commonest administered to a patient, in order to be kept up to the standard of 2500 to 3000 calories in twenty-four hours, means that the patient must take from 3 to 4 quarts (3000 to 4000 mils) in each twenty-four hours. Thus, if the milk is given every three hours, night and day, he must take 12 to 16 ounces (360 to 480 mils) at each feeding. If the interval is two hours, night and day, the amount at each feeding must be from 8 to 10 ounces (240 to 300 mils) at each feeding. Anyone who has attempted to force down the throats of patients, even only slightly ill with typhoid fever, continuous doses of milk, knows that it is an impossible task. In the first place, the patient rebels at the large amount of milk, if he be not too ill to rebel, and in the second place, it is not wise to awaken a patient every two hours during the day and night. This deficient food value of milk is one of the great objections to its exclusive use as a diet in typhoid fever. This can be overcome by reinforcing it with cream and milk-sugar, thereby increasing its food value so that the quantity administered

may be much less, and the food value greater than when plain milk is used. For instance, 1 quart (1000 mils) of milk, 750 calories; 4 ounces (120 mils) of cream, 240 calories; 4 ounces (124.4 Gms.) of milk-sugar, 240 calories, making a total of 1230 calories. The following table, frequently used by the author, may be used with very great satisfaction:

6 A.M.	6 ounces (180 mils) of reinforced milk...	250	calories.
7 A.M.	Soft egg, dish of cereal, 1 slice bread and butter	200	"
9 A.M.	6 ounces (180 mils) of reinforced milk...	250	"
11 A.M.	6 ounces (180 mils) of reinforced milk...	250	"
12 NOON.	Milk reinforced, 1 baked or mashed potato, 1 slice of bread and butter, or toast, coffee or tea	425	"
2 P.M.	6 ounces (180 mils) reinforced milk.....	250	"
4 P.M.	6 ounces (180 mils) of reinforced milk...	250	"
6 P.M.	Minced chicken, or junket, or soft part of 4 oysters	75	"
8 P.M.	6 ounces (180 mils) of reinforced milk...	250	"
		2200	"

This combination will allow the patient more than 2000 calories of food intake, and the amount will but little exceed 1 quart (1000 mils) of liquid. Broths are of little food value, but many patients are given these liquids, which are grateful, but which contain but little real nourishment. It is of slight avail to the patient if he is given a quart or so of broth; his desires are satisfied, and he is allowed to be underfed, thinking himself overfed. Milk, of course, can be given in the form of junket, oyster stews, and in many other ways. It can be flavored with coffee, salt, or any flavoring desired, in order to create a real appetite in the patient. If one desires to give a liquid diet to the patient because he is too ill to eat, as before stated, the main reliance must be placed upon milk. Reinforced milk will give sufficient nourishment without overburdening his stomach with liquid.

When the patient is able to eat, it is well to give him a diet consisting of foods other than milk. This lightens the tedium of an exhausting illness, helps to keep up the strength of the patient, and does not nauseate him by its routine sameness. For a number of years a liberal dietary has been used by many clinicians. There is an abundance of careful obser-

vation to show that patients fed on liberal diet are at least no more prone to complications, and surely become less toxic, than are those on liquid diets; and, indeed, statistics show figures in favor of a mixed and free diet. The author's practice is to give a diet in which reinforced milk forms a large element, but which contains a liberal diet besides the milk. The following routine is ordered, subject, of course, to any variations that seem necessary. The formula of the milk mixture which gives a total of 1500 calories can be used.

In this way the patient gets an abundance of food, is not worried by a monotonous dietary, and is allowed to rest during the night. A list of proper foods, taken from an article published by Frederick Shattuck, is found below:

Shattuck Diet. 1. Milk, hot or cold, with or without salt, diluted with lime-water, Apollinaris, or Vichy. Peptonized milk; cream and water (*i.e.,* less albumin), milk with white of egg, buttermilk, kumiss, matzoon, milk whey, milk with tea, coffee or cocoa.

2. Soups: beef, veal, chicken, tomato, potato, oyster, mutton, pea, bean, squash, carefully strained and thickened with rice, powdered arrowroot, flour, milk, cream, egg or barley.

3. Horlick's food, Mellin's food, malted milk.

4. Beef-juice.

5. Gruels: strained cornmeal, crackers, flour, barley-water, toast-water, albumin-water with lemon-juice.

6. Ice-cream.

7. Eggs, soft-boiled or raw; egg-nogg.

8. Finely minced lean meat; scraped beef, the soft part of raw oysters; soft crackers with milk or broth; soft puddings without raisins; soft toast, without crust; blanc mange, wine-jelly, apple-sauce and macaroni.

Ice-cream may be given in any quantity which does not cause nausea or diarrhea.

Care must be taken, however, in the use of such a diet as this, to see that quality is not sacrificed to quantity. It is very easy to satisfy the appetite of patients with typhoid fever, and, indeed, they are frequently without any appetite, so that one must, by the arrangement of the sort of diet shown above, make it quite possible to give enough in quantity as well as in quality.

Control of the Temperature. There can be no doubt from many observations that, while fever is but a symptom in the course of the disease of typhoid fever, it is a symptom which, by itself, can do harm. It is certainly true that the control of excessive fever removes one of the elements of grave danger in the disease. Therefore, the application of cold in one way or another is a necessary part in the treatment of any given case where the fever continues at 102½° F. (39.1° C.) or above.

Brand's method of cold plunging is still a favorite method of controlling the temperature at the hands of the writer. This needs for its application a portable tub of some character, and hence its use in a private house, except where the cost of treatment is not a question of importance, is impracticable. This is another argument in favor of the treatment of typhoid fever in a well-regulated hospital, where a tub is available and skilled help to move the patient in and out of the tub is at hand. The writer believes that the Brand method should be used as a routine procedure of hospital treatment. This belief is not founded on fanciful theories, but is the result of the good effects of a cold plunge upon patients who have been inefficiently sponged, or in whom the temperature has been disregarded. The routine method of the bath treatment is as follows:

The patient is plunged when the axillary temperature reaches 102½° F. (39.1° C.) or over, but not more than eight plunges are given in twenty-four hours. A tub is used which is large enough for the patient to lie full length with the shoulders and head slightly raised. The temperature of the bath varies with the reaction of the patient. Water at 80° F. (26.6° C.) is a good average temperature for the beginning of the bath. If the patient reacts well, the temperature of the water may be gradually lowered by the addition of ice to the bath. If he does not react well, the temperature may be raised by the gradual addition of warm water. A tub thermometer should be used, so as to make the observations accurate, and the nurse should not attempt to judge of the temperature of the bath water by testing with the hands. Of course, the water in the tub should be renewed for each individual bath. The tub is wheeled to the side of the bed,

the patient is stripped and lifted from the bed by three assistants, one taking the head and shoulders, the second the buttocks, and a third the feet and legs. For reasons of modesty, the patient should be kept covered with a sheet or a very light blanket while being moved. After the patient is in the tub, the limbs and body are rubbed briskly by the nurses, so as to encourage the peripheral circulation. It is essential to see that an ice-cap is kept upon the head of the patient while in the bath, in which he remains for a period of from fifteen to twenty minutes.

Before lifting him from the bath, the bed is arranged with a mackintosh covered with a light blanket, upon which the patient is laid and rubbed until he is comfortably warm. Vigorous rubbing should be restricted to the torso and limbs, as no massage of the abdomen is permissible. Half an hour to three-quarters of an hour after the bath the temperature is again taken and recorded. Another bath is given when the temperature of the patient reaches 102½° F. (39.1° C.) again, provided that an interval of three hours has elapsed since the beginning of the previous bath.

The effect of tubbing on the appearance and feelings of the patient is usually unpleasant. He shivers, the fingers and lips may become blue, and the radial pulse is difficult to appreciate. This difficulty in feeling the pulse, however, is almost without exception the result of muscular tremor, which makes it difficult to recognize the pulse. If the stethoscope be placed over the heart, however, the beats will be found to be regular and strong, and not unduly hurried. Of course, if the heart's action becomes feeble and irregular (rarely the case) and the patient goes into collapse, he must be removed from the bath immediately, and at the next occasion the bath water must be of a higher degree of temperature than the one in which the collapse occurred. If the bath has been beneficial, the temperature after the bath will be from one to three degrees lower than before the bath was given; the pulse will become slower, and, as a rule, the patient falls into a quiet, rational sleep. The contraindications to the bath are intestinal perforation, intestinal hemorrhage and collapse. The next best method of applying cold in this disease is the form of a *cold affusion.*

To give this treatment properly, the bed should be covered with a rubber blanket large enough to extend over the head, foot and sides of the bed at least one foot. Each corner of this blanket is then tied, bringing a part of its side and ends together, to form a trough in which the patient lies. Water of the required temperature is then allowed to drip upon the bare skin of the patient, and, collecting in the blanket, makes a very respectable sort of bath. The water is then sponged from the blanket, and the patient rubbed, and covered with a light blanket.

Another fair substitute for the Brand hydrotherapy is a *sponge bath* given with cold water, the patient being stripped and the parts rubbed. If given after the manner of a cleansing bath it will be of little avail in lowering the temperature of the body. In order to do this, which is only one of the objects and the results of the bath treatment, the water must be cold enough and the amount large enough to attain those objects.

The other object of hydrotherapy, aside from lowering of the temperature of the body, is stimulation of the heart's action and that of the nervous system. This is a usual result. Elimination of toxins through increased urinary output is another one of the good results of the cold treatment. A method of reducing temperature has been suggested by Williams, in which the body of the patient is covered with gauze or other thin material, over which is sprinkled tepid water. The body is then fanned, either with a hand fan or by an electric fan, and the evaporation of the water thus produced cools the body. The writer has not used this method, but he doubts whether it would have all the good results of a cold plunge.

Water should be given in abundance, and if a veritable polyuria can be obtained by this means, much value will follow.

Regulation of the bowel movements is a most necessary part of the treatment. It is a well-known fact that diarrhea is recognized as a common symptom of the disease. On the other hand, constipation is sometimes present from the beginning to the end of the disease. It is a custom among practitioners, when a case of suspicious typhoid fever presents

itself, to give a preliminary dose of calomel. This is frequently taught in the various schools of medicine. In the very earliest days of the disease such a routine measure is permissible, but when there is any doubt as to the time the disease has existed, it is probably wrong to give a purgative indiscriminately. In the second or third week a purgative is *harmful* and not helpful. Therefore, if during the first few days, up to perhaps the third or fourth day, there appears to be any retention of material in the bowel which is causing irritation, small doses of calomel are permissible, but it should not be given as a routine. During the active course of the disease, calomel should *not* be given, for there is too much danger of causing hemorrhage or perforation by a free purge.

Diarrhea calls for treatment when there are more than three or four bowel movements in twenty-four hours. It is treated according to the cause. If it appears to be the result of ingested food, the amount of food is reduced, or the offending kind of food withdrawn. If it is the result of the toxic state, this is especially attended to by reduction (by general means) of that condition, as much as possible, in a way to be spoken of later.

If it seems to be the result of retained material, flushing of the lower bowel will be of value. Care must be taken to see that the diarrhea is not a symptom of obstruction, due to large fecal masses in the lower bowel. This complication can be easily recognized by rectal examination of the bowel with a gloved finger.

The diarrhea may be consequent to irritation by intestinal ulcers, and this factor is, perhaps, the commonest cause.

The two drugs which have been of the greatest use to the writer are bismuth and phenol salicylate (salol), given in doses of efficient size, combined with chalk mixture. Such a mixture as the following is valuable:

℞ Bismuthi subnitratis ..... 16 (246.8 gr.).
Phenolis salicylatis ..... 8 (123.4 gr.).
Misturæ cretæ compositi ..... 120 (4 f℥).

M. S.: One teaspoonful (4 mils) every two hours.

Useful Drugs. As stated early in this article, there is no specific drug for typhoid fever. Therefore, the use of drugs must be entirely symptomatic. If there are any drugs which

are to be used in a routine method, those are the mineral acids and intestinal antiseptics, and the urinary antiseptics. The author is fond of using a combination such as follows:

℞ Acidi hydrochlorici diluti ..... 16 (246.8 gr.).
Tincturæ nucis vomicæ ..... 8 (123.4 gr.).
Tincturæ gentianæ compositi .... 120 (4 f℥).
M. S.: One teaspoonful (4 mils) every three hours.

When the tongue becomes coated and thickly furred, and entire disgust for food appears, this acid alcoholic mixture seems to be of decided value in ameliorating the symptoms.

Intestinal antiseptics have appeared to me to be of value in preventing or lessening the tendency to diarrhea and tympany.

Phenol salicylate (salol) given in doses of 5 grains (0.325 Gms.) every four hours seems to be of value. Certainly the fetid character of the stools is frequently lessened, and the meteorism becomes less after the use of this drug. Another intestinal antiseptic, or one or the other combinations, may also be of value. Turpentine is of decided utility when there is much meteorism, and when the tongue is brown and hard and dry. It may be given as an emulsion.

℞ Olei terebinthinæ ..... 16 (246.8 gr.).
Mucil. acaciæ,
Aq. menthæ pip., q. s. .....āā 120 (4 f℥).
Ft. emulsio.
S.: One teaspoonful (4 mils) every three hours.

Alcohol. This drug is still used by some practitioners in a routine way. This is a mistake, for alcohol seems to be of value in certain conditions, such as an entire lack of ability to take food. But to use it as an actual heart stimulant, is to use it in a manner which is harmful.

As a food it may be used in the form of whisky or brandy in doses of 2 or 3 ounces (60 or 90 mils) every twenty-four hours. To use it in larger doses is to do harm. The custom of using 6 to 8 ounces (180 to 240 mils) of alcohol in twenty-four hours is pernicious, for it weakens the heart and lowers the vitality.

Caffein can be used as a cardiac stimulant, either in the form of the alkaloid, caffein, or hypodermically, as caffein and sodium benzoate.

Hexamethylenamin should be resorted to as a routine in every case. It has been shown by Gwyn and others that the consistent use of this drug throughout the disease tends to keep the urine free from typhoid bacilli.

Strychnin should be used in doses of $\frac{1}{30}$ grain (0.002 Gm.) at intervals varying from eight to three hours. In my experience it is unwise to use strychnin in a routine manner in doses greater than just specified every three hours. This makes approximately $\frac{1}{4}$ grain (0.016 Gm.) in twenty-four hours, and more than this is often accompanied by signs of physiologic action of the drug.

It has been shown lately that in certain cases of fever digitalis appears to have exactly the same physiologic action as when used in cases of cardiac disease, or when used in normal individuals. It is my personal experience, however, that digitalis by the mouth or hypodermically, when employed in cases of typhoid fever with a rapid heart action and failing circulation, is practically useless. The amount of toxin present in the blood and affecting the heart-muscle seems to render the drug entirely useless. It seems much better to depend upon caffein and ammonia.

COMPLICATIONS.

I wish to add emphasis to the word *early* in the sentence used under the heading of treatment. Some complications, such as perforation of the bowel and laryngeal perichondritis, are practically always fatal if not recognized early. Others, such as a nephritis or cardiac complications, acute dilatation of the stomach and phlebitis are treated much more successfully when recognized in the early stages of their development. If left until the full picture is present they may be fatal, when early recognition may avert a serious or fatal issue.

Perforation of the Bowel. This is a surgical complication for which the only practical cure is a laparotomy. Every pain in the abdomen of a patient with typhoid fever does not mean that the patient has a perforation of the bowel, but every abdominal pain should have the most serious and repeated attention. If we are to escape disaster, we must do several things: *First,* instruct the nurse to report at once if the patient complains of abdominal pain; *second,* see the

patient immediately, whether the message comes during the day or night, and then go about making a diagnosis without undue delay. With the patient's chest and abdomen bared, the chest should be examined first. It must always be remembered that a complicating pneumonia, with pleurisy as its earliest symptom, frequently will simulate abdominal inflammation by the pain referred to the abdomen. If this be the case, always keep in mind that, with a careful chest examination in such accidents, fewer cases of pleurisy and pneumonia will be mistakenly submitted to abdominal operations. When one is sure that the chest is in normal condition, then the abdomen is to be carefully examined. The symptoms and physical signs characteristic of perforation are sudden, severe abdominal pain, with local tenderness and resistance. If these symptoms persist, and if, in addition, the pulse becomes more rapid, the temperature gradually increases, and an increase in the number of leucocytes occurs, there is sufficient reason for a laparotomy, and a surgeon should be summoned immediately. Indeed, it is well to have the surgeon see the case at the very onset of such an alarming symptom-complex. In certain instances the symptoms and physical signs are so characteristic that a positive diagnosis can be positively made at the very first visit. As intimated above, one must carefully differentiate pain due to flatulence or to appendicitis from this condition, because an operation in the midst of a case of typhoid fever is a serious matter, but delay in the operation in cases of perforation is fatal to the well-being of the patient. After careful examination we must operate if signs warrant it. If we wait until abdominal distention, disappearance of liver dullness, rapid pulse and fall of temperature are present, we have waited too long. The operation has the best chance of curing the patient if done in the first few hours after the appearance of symptoms. Be as sure of the diagnosis as possible, and then operate.

A routine blood examination is of great help in the treatment of typhoid fever. A full count made early gives data which is of value. This count should be repeated every week at least, and every day, or oftener, in certain conditions. The early count gives us a guide to the condition of the blood.

Any increase of the leucocytes, or decrease of the hemoglobin and erythrocytes, will then be interpreted in their proper way.

Intestinal Hemorrhage. Hemorrhage from the bowel is difficult to treat, for the reason that we lack means of controlling the bleeding. Each stool should be carefully examined by the nurse in charge for the appearance of blood, and if blood is found, either in large or in small quantities, all food should be stopped at once. The physician should then be informed, and should give such orders as seems proper in each case. All bathing and sponging should cease. The patient should not be told that he has had a hemorrhage. Physical and mental rest are most important. If there is evidence of a large hemorrhage, either in the amount of blood passed or in the physical signs, a hypodermic of ⅛ grain (0.008 Gm.) of morphin should at once be ordered. The foot of the bed should be raised and the patient left strictly alone. If the hemorrhage is repeated, the patient should be given 10 grains (0.65 Gm.) of calcium lactate every three hours, or a pill consisting of ½ grain (0.032 Gm.) of acetate of lead, and ½ grain (0.032 Gm.) of powdered opium should be given every three hours, care being taken to watch for symptoms indicating the general effect of the opium. The patient should not be narcotized. If the patient develops hemorrhagic tendencies, hypodermic injections of 10 mils (2.66 f3) of normal horse serum, repeated every three hours, should be given regularly. If there is much failure of circulation, the arms and legs should be freely bandaged, beginning at the hands and feet respectively, and the foot of the bed should be raised. If practicable, a Crile rubber compression apparatus should be used.

The question as to whether cardiac stimulants and intravenous injections of normal salt solution shall be given or withheld is always a very difficult one to solve.

The rapid pulse and weakness, with a tendency to faintness, is one of the normal results of the large hemorrhage. This should not be interfered with, unless the degree of cardiac failure is so extensive that it threatens life. To stimulate the circulation with drugs, and then to overfill the vessels with normal salt solution is to run grave risk of causing a repetition of the hemorrhage which may be fatal. Cer-

tainly elevation of the foot of the bed, bandaging and morphin must be tried first.

If drugs are used, hypodermics of strychnin, caffein and camphor may be used. Digitalis, as already stated, is useless. Whisky, brandy or champagne may be tried in small doses. Turpentine appears to relieve the flatulence, and, perhaps, is of value for its effect upon the bleeding surface. Water may be given by the mouth if the patient can retain this better. Intravenous injection of salt solution or hypodermoclysis may be tried. Not more than a pint (480 mils) of liquid should be injected by either method, but this should be repeated as necessity demands.

There is no rule which will cover all cases as to when these means are to be used, but each case must be judged by itself. This, to my mind, is one of the most difficult points upon which to give judgment. During the first twenty-four hours albumin-water, the white of an egg dissolved in a glass of water, may be given every three hours. This will give adequate nourishment, and if it be sweetened with 1 teaspoonful (4 mils) of milk-sugar, it will contain more nourishment, and will not cause any irritation of the bleeding intestinal tract. This nourishment may be given for forty-eight hours. After forty-eight hours with no return of bleeding, food may be gradually resumed, first liquids, egg-water reinforced by milk, then peptonized milk, gradually toast, and at the end of five or six days the food which was being taken at the beginning of the hemorrhage.

Acute Dilatation of the Stomach. This condition is not so common as a complication of typhoid fever as it is of some other diseases, such as pneumonia, but it does occur, and should be recognized. It resembles obstruction of the bowels, comes on suddenly, with signs of collapse and vomiting of extremely large quantities of foul-smelling material. The greatly enlarged stomach can be made out occupying the entire epigastrium, and even the upper half of the abdomen may be filled by this greatly distended stomach.

The treatment for acute gastric dilatation is to wash out the stomach with large quantities of salt solution, to elevate the foot of the bed, and to place the patient either on the right side or on the face.

Because of the great resemblance of this condition to intestinal obstruction, one may be tempted to open the abdomen. This is fatal, and where there is the least doubt in the mind of the physician, the stomach should always be emptied by a stomach-tube, as a means of certain diagnosis.

Laryngitis. This is a rather frequent complication of typhoid fever. It is recognized by a more or less hoarse, unproductive cough, and examination of the larynx shows a reddened mucous membrane, both of the general mucosa and of the vocal cords. Its treatment consists of the application of cold to the neck in the form of ice-bags or cold compresses, and of inhalation of steam.

Bronchitis. A relatively active tracheobronchitis is present in a more or less severe grade in practically all cases of typhoid fever, and, if severe, and especially when it affects the smaller bronchi, there is necessity for treatment. If there is not much expectoration, citrate of potassium is of value. This can be combined with ordinary compound licorice mixture. This, as a rule, contains enough opium to control the cough, and if it does not, paregoric may be added in appropriate doses. If the patient is restless and nervous, the combination of bromide of ammonia and bromide of potassium is of value. Such a mixture as the following is useful:

℞ Potassii citratis ..... 8 (123.4 gr.).
Potassii bromidi ..... 16 (246.8 gr.).
Misturæ glycyrrhizæ comp. q. s. 120 (4 f℥).

M. S.: One teaspoonful (4 mils) every three hours.

Laryngeal Perichondritis. This complication is rare, but needs immediate treatment when it occurs. It is preceded first by hoarseness. Examination of the larynx will show a redness of the mucous membrane. As the condition advances, the tissues over the larynx become swollen, edematous and exquisitely tender. If fluctuation occurs, the abscess may be incised. If stridor occurs and great difficulty in breathing supervenes, then the patient may develop a sudden edema of the larynx. Everything must be kept in readiness for a tracheotomy, so that this simple operation may be done at an instant's notice, in order to save life.

Periostitis and Ostitis. These complications occur late in the course of the disease, or after convalescence has set in.

Here the great error in diagnosis is to consider the painful swelling as due to rheumatism, and an error of this sort may delay proper treatment until severe lesions occur, which may readily be fatal.

Early recognition and early operation are of the greatest importance, and if operation is done early there will be little danger of a serious outcome.

Pleurisy. Pleurisy is recognized by a sudden pain in the chest, aggravated by taking a deep breath, by rapid, painful breathing, and by the physical signs of a friction rub. If the pain is extreme, opium should be used and the chest strapped. This complication is frequently the forerunner of a true pneumonia. Occasionally a pleural effusion contaminated by typhoid bacilli develops, as in the rare case reported by Pepper.

Pneumonia. This complication may be an ordinary acute croupous pneumonia, the result of infection by the pneumococcus. It is occasionally due to the implantation of the typhoid bacillus, and then results a true typhoid pneumonia. Its symptoms and treatment are no different from the treatment of pneumonia under ordinary circumstances. The routine treatment of the typhoid fever, not excepting the tub baths, need not be interrupted. Digitalis here may be used with apparently more effect than when the heart becomes weak simply from the typhoid poison alone.

Hypostatic congestion of the lungs is very likely to occur in the more severe cases of typhoid fever. The lungs should be daily examined, and if there are any signs of congestion at the bases, the position of the patient should be changed constantly, so as to relieve this congestion. Usually caffein, strychnin and ammonia are necessary under these conditions.

Cardiac Weakness. Cardiac weakness is the result of the poison of the disease. It is due to the effect of the poison on the cardiac muscle. It must be combated by the general tonic methods as described under general treatment.

Endocarditis and Pericarditis. Both endo- and pericarditis occur as the result of infection of the endocardium and the pericardium by the typhoid bacillus They can, of course, be recognized by careful examination of the heart itself. Extreme quiet is necessary. Application of cold is of value.

Digitalis should not be used under these circumstances. It causes a more forcible heart action, and may be harmful.

Phlebitis. This is characterized by pain and tenderness along the line of a superficial vein. Any vein may become inflamed, the most usual one affected being the left external saphenous. In addition to the pain and tenderness along the vein, there is frequently redness, the hardened vein can be felt to roll under the fingers, and there is also edema of the foot and leg on the affected side. This edema first appears about the inner maleolus. With a rise of temperature and leucocytosis, it is wise to examine this part of the leg, because often this is the first point to show disturbance. The whole leg, from foot to groin, should be bandaged lightly, preferably with a gauze bandage, and elevated upon pillows so that the foot should be slightly higher than the pelvis. Applications of a saturated solution of magnesium sulphate gives relief to the pain, which is more or less severe. This is more cleanly than, and just as useful as, the usual use of ointments. If the pain is so great as to cause much distress or wakefulness, opium in some form can be used.

Nephritis. Albuminuria with a few tube-casts is a constant accompaniment of practically all cases of typhoid fever of any severity. This need not give any concern, and does not call for any special treatment. However, frequent examinations of the amount and character of the urine should be made. If the amount decreases, more water should be given in order to increase the output.

If a true nephritis occurs, as indicated by suppression of the urine and the presence of blood, blood-casts, and dark, granular casts, the diet must be restricted to the most bland articles, and water in large quantities is to be given.

Cystitis. Is an occasional complication, and is best treated by the use of hexamethylenamin.

Delirium. This is a symptom common to almost all cases of typhoid fever, but sometimes it needs special treatment. The bromids can be used with benefit, cold to the head is of value, and merely the voice of the nurse, calming words, or the stroke of a hand will occasionally do much to quiet the delirious patient. Morphin hypodermically, alone or combined with hyoscin or scopolamin, will sometimes be neces-

sary to give the patient rest. The low, muttering delirium, with picking at the bed-clothing, like the ordinary delirium of the disease, is an indication of the toxemia. It is best combated by elimination, which can be obtained from the ingestion of water, either by the mouth or administered through hypodermyolysis or by intravenous injection of normal salt solution. Actual insanity occurs sometimes as a post-febrile condition. This is doubtless toxic and is due to the weak condition of the patient causing a cerebral disturbance. It is best combated by increasing the nourishment of the patient. If the delirium is extremely wild, then the use of bromids, and, if necessary, opium is indicated.

Physical restraint by means of restraining sheets, a simple sheet folded into a strip 12 or 18 inches (30.4 or 45.7 cm.) in breadth, passed over the chest, and fastened to the bed rails, is helpful. The patient may be allowed to move his hands and feet, but the pull of the sheet over the chest often causes the patient to cease his efforts of attempting to get up. Cuffs, restraining straps to restrain the legs and arms, and a regular strait-jacket should not be used. It is better that the patient be quieted by some narcotic than allowed to exhaust himself with the physical exertion he exerts when subjected to mechanical restraints of this sort.

Meningitis. This is a rare complication, but occasionally demands attention. Meningisimus, however, occurs, which in many ways resembles a true meningitis, and both meningitis and meningisimus are treated well by spinal puncture. This can be repeated every twenty-four or forty-eight hours with nothing but good results. Bromids may be used, also hyoscin. Morphin, for some reason, apparently precipitates a coma in certain cases; it should be used with caution, in small doses, and better in combination with hyoscin than alone.

Furunculosis. This is a post-febrile complication, and gives rise to a low grade of fever and decided emaciation. Early evacuation of the pus is important. An autogenous vaccine, made from a culture of the contents of the furuncle, is often of the greatest value.

Cholecystitis. Frequently the gall-bladder becomes inflamed during an attack of typhoid fever, giving rise to pain, tenderness, and resistance in the region of that organ. The

symptoms of this local condition are increased fever and leucocytosis. The situation of the pain over the gall-bladder itself allows a probable differentiation between this condition and appendicitis. The treatment of cholecystitis in all but rare cases is one of watchfulness and treatment of the symptoms—cold compresses or an ice-bag over the inflamed gall-bladder, with perfect rest and the administration of hexamethylenamin are the best methods to pursue in the vast majority of cases. Occasionally, however, there are cases in which the inflammation goes on to suppuration, and we are confronted with a condition of much danger, suppurative cholecystitis. If the fever becomes intermittent, if the leucocytes rise rapidly, and if a mass appears over the gall-bladder, it is best to call a careful, conservative surgeon in consultation. A laparotomy should certainly be performed if one is satisfied as to the existence of suppuration.

Appendicitis. The appendix is frequently the seat of inflammation during typhoid fever, but usually the process is simple in type; the condition is not of very grave moment, and disappears with the subsidence of the typhoid infection. Occasionally, however, a typhoid ulcer invades the appendix, and may give rise to perforation. Also, occasionally the appendix becomes very acutely inflamed, and ulcerative or suppurative appendicitis is present. These latter conditions can be diagnosed by increasing circumscribed tenderness and resistance of the belly, and by increasing leucocytosis with fever and distress of the patient. When appendicitis occurs as a complication of typhoid, the same rule prevailing when it appears in a patient in good health cannot be safely observed. In health an operation should be done at the earliest moment, but the dangers of an operation during the course of typhoid fever are greater than the danger of waiting in the usual case. The mild cases must be treated symptomatically with cold, starvation and opium to relieve the pain. If, however, the case goes on to suppuration, an operation must be done at the earliest possible moment.

The rule to follow, then, is to operate when the danger from the local appendicitis appears greater than the danger from an operation. In health, I repeat, given a good surgeon,

the dangers of an operation are *nil* as compared with the potential dangers of an appendicular inflammation.

Convalescence. The patient's convalescence will be shorter if the food has been kept up to the maximum in amount during the stage of active fever. As a rule, one week after a patient is free from fever night and morning, he can be allowed to sit up in bed. In two weeks he can be out of bed and walk about. Care not to overload the stomach with food is one of the most necessary rules of convalescence. Let the food be mixed, of almost any reasonable kind, but it must be moderate in quantity.

Exercise must be very gradually undertaken, the first act sitting up in bed, next in a chair beside the bed, then short walks, and at the end of two weeks, walks which will be of considerable length.

Typhoid spine, which is probably a periarthritis, often gives rise to an extreme amount of pain and a semi-invalidism for a long series of weeks. Massage, occasionally strapping, and sometimes a light steel brace are a help. The use of potassium iodid is of value.

Use of Vaccines. The prophylactic use of vaccines in typhoid fever has been abundantly proven, as shown by their employment in the American army and in the armies of Europe now at war. The method of use employed in America is to use the bodies of typhoid bacilli killed at 53° C. (127.4° F.); 500,000,000 of these are injected hypodermically; in a period of ten days 100,000,000 are injected, as the initial dose, and ten days later another 100,000,000. Frequently there is a local induration which is red, swollen and edematous. Occasionally there is a feeling of malaise with rise of temperature after each injection, but usually the post-vaccinal reaction is so mild that the patient does not have to keep his bed. Infrequently the reaction is more severe, and the temperature rises as high as 102½° F. (39° C.), and in such cases the patient is likely to be indisposed for several days.

The therapeutic use of vaccines is still on trial. Many reports are published appearing to prove that cases treated with vaccines of the typhoid bacillus do well, but, on the contrary, other reports throw doubt on the actual value of this method of treatment. On the whole, the effect induced by

the foreign protein may be more deleterious than that of the disease it is meant to combat. Apparently there are no bad results reported. In this connection several articles recently published would indicate that any foreign protein has an effect on the cases of typhoid fever in which they have been used intravenously similar to the effect of the specific typhoid vaccine.

PARATYPHOID FEVER.

This disease closely resembles infection by the true typhoid bacillus. Indeed, the paratyphoid bacillus *a* and the paratyphoid bacillus *b* are but varieties of the typhoid bacillus. The disease can be distinguished from true typhoid by the fact that the blood-serum of the patient will not agglutinate typhoid bacilli, but will agglutinate either the paratyphoid *a* or *b* bacteria. The actual specific diagnosis, however, can be made by blood-cultures, or cultures from the stool or urine.

The symptoms of paratyphoid are essentially those of typhoid fever.

The diagnosis, prophylaxis and treatment differ in no way from that of typhoid fever. The reader is referred for details to the article on the treatment of typhoid fever (page 7).

CROUPOUS PNEUMONIA.

Croupous pneumonia, or lobar pneumonia, is due to infection by the pneumococcus, a micro-organism usually found in the mouth and in the air-passages of healthy persons, and prone to become virulent under certain circumstances. The exact reason why these organisms are virulent under certain conditions and not under other, and even similar ones, is not known. The symptoms present in the usual case of pneumonia are an initial chill of more or less severity; fever, the temperature rising rapidly to 103° or 104° F. (39.4° or 40° C.), with dyspnea and pain in the chest, aggravated by taking a long breath. The degree and continuance of the pain depends upon the extent of the pleuritic lesion incident to the individual case. As the case progresses, the symptoms vary with the degree of pulmonary consolidation and with the

degree of toxicity in the case in question. Occasionally the patient is extremely ill, with high fever, rapid cardiac action, delirium, and every evidence of extreme toxicity, while the amount of pulmonary implication is comparatively small. In such instances there is a true pneumococcemia.

On the other hand, the size of the pneumonic area may be very great, the dyspnea and cyanosis extreme on account of the extensive solidification, and yet the subject shows but moderate toxemia.

The disease is, however, a true pneumococcemia, as proved by the fact that the micro-organisms have been found in the blood-stream, and also by the fact that organs other than the lungs become inflamed by the local inflammation excited by the pneumococcus, for instance, in the brain and spinal cord.

The physical signs of the condition consist of dullness over the affected area of the lung; at first distant breathing with crepitant râles; and within twenty-four hours, depending largely upon the resistance of the patient and upon the degree of infection, in the usual case, bronchial breathing. The degree of further progress varies, usually one single lobe is pneumonic, and this is followed by invasion usually of an adjoining lobe. There is practically always a pleurisy surrounding the lobe affected. This may, or may not, give rise to an exudate, which either remains serous or becomes purulent. Death usually occurs as the result of failure of the circulation, which may be in turn due either to the existing septicemia or to dilatation of the right heart, the result of the pulmonary process.

The problem of treatment, then, is, *first,* the use of specific sera or specific drugs; and, *second,* the use of other than specific measures.

SPECIFIC TREATMENT.

When sera were first used as a treatment for bacterial diseases, and when diphtheria and tetanus came under the ban of the antitoxic sera, it was hoped, and, indeed, expected that the pneumococcus would give as efficient a serum as the diphtheria and tetanus bacilli yielded. Until recently this hope has not been fulfilled. The mortality in a large number of cases of pneumonia treated by antipneumococcic serum

was no lower than that of those treated by ordinary expectant means. Within the last three years, however, Cole, of the Rockefeller Institute, has put the specific treatment of pneumonia upon what appears to be a firm basis. As yet, unfortunately, the methods proposed are too cumbersome to make the method practicable for the use of anyone who has not access to a well-equipped hospital, and to a laboratory in which the proper bacteriologic examinations can be made. Philadelphia, through the Board of Health, now gives opportunity to physicians to use this serum.

Cole in two addresses, one before the State Medical Society of Pennsylvania, and the other before the Congress of American Physicians and Surgeons in Washington, makes statements about the specific treatment of pneumonia, of which the following is an abstract:

In the first place, pneumonia, as recognized by the clinician, is not the result of an infection due to identical micro-organisms. It has been discovered that pneumococci can be divided into groups, the characteristics of the members of one group differing widely from the peculiarities of those of the other groups, and then, again, that about 5 per cent. of the cases diagnosed as pneumonia are due to infections by other germs, such as the staphylococcus, the streptococcus and Friedländer's bacillus.

In order to identify the various groups of pneumococci, the following method is used: A small portion of the sputum coughed up from the lung is injected into the peritoneal cavity of a mouse. After the micro-organisms have developed, the peritoneal cavity is washed out with salt solution, and the washing is then centrifugalized for a short time to remove leucocytes and other animal cells. To a small portion of this suspension, immune sera of types 1 and 2 are added. If agglutination occurs in either mixture, we know that we are dealing with a type of micro-organism corresponding to that particular serum. If no agglutination occurs, the bacteria are types 3 or 4. Type 3 bacteria can usually be detected by direct examination of the peritoneal exudate. The micro-organism possesses larger capsules than the others, and in animals produces a very stiff mucous exudate. Then there are a number of germs which do not fall into any of these

groups, but differ among themselves. This group has been called type 4. It is, therefore, seen that pneumococci can be grouped into four types, of which both types 1 and 2 produce in animals a protective serum, while types 3 and 4 do not produce protective serum. The immune serum of types 1 and 2 has been obtained from horses. Its method of production is that commonly employed in producing other therapeutic sera. Type 1 serum has a very great strength. The serum obtained against type 2 is of less potency. Very recently the New York Board of Health has been able to produce a serum against type 3 which, while it is of no value as a curative agent, shows that it may be possible hereafter to produce a serum valuable in infection from this particular micro-organism. The reason that sera, which heretofore have been employed as a curative agent, have been ineffective, is because the sera have not been produced with a knowledge that these various types of pneumococci produce sera which are curative only against germs of their own type.

After the observation of 432 cases of pneumonia at the Rockefeller Hospital and at the Pennsylvania Hospital, in Philadelphia, it has been discovered that approximately one-third of the cases of pneumonia are due to infections of type 1, one-third to infections of type 2, and the other third to infection of types 3 and 4. The mortality of the cases infected by type 1 and not treated by immune sera is about 25 per cent.; those of type 2 about 29 per cent.; those of type 3, 45 per cent., and those of type 4, 12 per cent.

The results of the treatment with type 1 serum are encouraging. The number so treated is not large, 72 cases, treated by type 1 serum, with 6 deaths, a mortality of 8 per cent. This is a distinct gain over the mortality of 25 per cent. when not treated with serum.

The method of treatment is as follows: As soon as a pneumonia patient is admitted to the hospital a ½ mil (8 *m.*) of serum is given, in order to test the sensitiveness of the patient and to desensitize the subject if possible. Then when the type of bacteria is discovered, 80 mils (2.6 f℥) of serum, are injected into the vein of the individual, and this procedure is repeated every twelve hours until the temperature and pulse rate fall. Following the injection a more

or less severe reaction is set up, as evidenced by rapid pulse, high temperature, and frequently a chill. These reactions, however, in the Rockefeller cases have not been severe. A large number of patients receiving this serum suffered from serum sickness after a week or ten days. These symptoms are distressing, but not dangerous, and in more than 100 cases treated at the Rockefeller Institute there were no serious results.

A second form of specific treatment is by the use of chemicals. Several years ago Morganroth undertook to study the action of quinin on pneumonia, and also investigated the effects exerted in the injection by a number of derivatives of the drug. He found that quinin is a specific bactericide against the pneumococcus, but that its action in this direction is very slight. He found, furthermore, that one of the cinchona derivatives, known as ethylhydrocuprein, possesses a bactericidal action against pneumococci in a very high degree, but, unfortunately, it is also a very toxic drug, and there is a close correspondence between the curative and the lethal doses, so that as yet it is scarcely a proper drug to use promiscuously. Another particular action of this derivative of quinin is that it makes the micro-organisms acquire a so-called fast quality, owing to which they become entirely resistant to the action of the drug, and are not affected thereby.

It is to be hoped that before a great time has passed a serum made under the Rockefeller rules will be put upon the market, and will be used freely by practising physicians, so that at least one form of the disease may have an actual specific cure for it.

TREATMENT BY OTHER THAN SPECIFIC METHODS.

Six essentials of treatment stand out as applicable to practically every case of pneumonia, aside from which there are many other helpful details to be carried out:

1. Early recognition of the case.
2. Absolute rest.
3. Abundance of fresh air.
4. Proper amount of good food.

5. Constant watchfulness.
6. Administration of proper drugs at proper times.

Early Recognition of Pneumonia. It is sometimes impossible, and always difficult, to make a diagnosis of pneumonia during the first twenty-four hours of the attack, but whenever the symptoms and physical signs in a given case are suggestive of pneumonia, the patient should be put upon whatever routine treatment is decided upon. Failure to appreciate the probability of an attack being one of true pneumonia may be followed by orders which will allow the patient to be up and about prematurely, and thus to become exhausted, and so subjected to a more severe attack than otherwise would have developed. The essentials as outlined are rest, fresh air, proper dietary and watchfulness.

Rest. Mental as well as physical rest must be absolute, and all unnecessary movement must be interdicted by the physician. The patient should not rise for urination or defecation, to which end a bed-pan must be used. Visitors in the sick-room should be distinctly limited, and, it is better, if the patient does not become restless under the restriction, to allow no visitors at all. On the other hand, if the presence of the wife or husband, or any other person soothes the patient, it will be best for that individual to be admitted to the sick-room. But care must be taken that the patient's room is not made a visiting place for all friends and relatives. In hospital wards where visiting is not controlled as carefully as in private homes, I think it is quite certain that patients affected with pneumonia, and, indeed, with any other serious condition, are not quite so well after visits have been made. The rest should continue several days after convalescence has begun. One must remember that the majority of uncomplicated croupous pneumonia cases get well after a sudden crisis, and that the remainder recover after a lysis of three or four days' duration.

For a week or ten days the patient has been extremely ill, and all his forces are weakened. Therefore, extreme care must be taken to avoid any unnecessary strain for several days. After the temperature has remained normal about one week, provided that the patient seems well otherwise, he may be allowed to sit up, at first with a back-rest, in bed, and later in an easy chair.

Fresh Air. This means pure, unheated, unbreathed air, admitted into the room through wide-open windows. A room with two windows opposite each other is best, so that a cross draft may be formed, thus keeping the air in the room constantly changed. The patient need not be placed in the draft, although, except for discomfort, it is a matter of indifference whether a draft blows over the sick one, provided that he be well protected with clothing so that the body will not be chilled. If it is practicable, it is still better for the patient to be on an open porch, protected so that the wind and storm will not drive over his body. To the writer's mind the direction to keep the temperature of the room between 60° and 70° F. (15.5° and 21.1° C.) is a mistake. The temperature of the air in the room should be that of the outside air; in cold weather the patient should have blankets both over him and under him, and be clothed in a cap and warm, loose underclothing, opened at the back to facilitate easy removal. Thick mittens on the hands are comforting, if the patient desires to have his hands outside the bed-covers. The nurse should be warmly clad with a soft, thick sweater; her feet should be protected with proper shoes, and her legs and ankles with warm underclothing. If the patient's bed-clothing has to be changed, or if the body has to be exposed, then, of course, the windows in the room must be closed, and, if practicable, heat turned on in the room. It is proper to have one of two arrangements: either the patient should be treated on a porch connecting with a room into which the bed can be rolled when changes are necessary, or two adjoining rooms should be used, the one in which the patient remains with the windows wide open as directed, the other to be kept warm for changing the patient's clothing. The time which the patient will spend in the protected room will depend largely upon the patient himself. Time and again one who has been out on the porch or in an open room with the temperature far below freezing, when brought into the warmer room, which has been closed for the purpose of changing the patient, will make the request to be put into the open room, or on the porch as soon as possible.

If inquiry is made of the patients they will almost without exception declare themselves more comfortable in the open

porch or room. While it has seemed that pneumonia subjects do better in open cold rooms than when the weather is warm, it is not so much the temperature as it is the fresh air which is beneficial.

If a patient is put into a cold room, or on a cold porch, and kept there without free circulation of air, he will suffer about as much as if he were in a closed warm room. Open the windows, and at once the patient will be more comfortable, whether the air be cold or warm. This rule of keeping the air fresh and in full circulation holds whether it is clear and dry or rainy and wet. Protect the room and the bed from the storm by means of screens, but allow free circulation of the air.

These opinions may seem too radical, especially as to the opinions expressed regarding the cold and drafts. It is based upon the observation of many cases in hospital and private practice, and there can be no question as to the good results coming from the fresh air. Protect the patient, and neither cold, drafts, nor dampness will harm him.

Food. The food necessary to a pneumonia patient should be nourishing, and of not too great quantity. It should be of a character which will not undergo fermentation in the intestines any more than is possible. The bowels should be regularly moved, so as to prevent the collection of fecal material, and consequent intestinal distention.

Food should be stopped or reduced to a minimum when abdominal distention becomes serious.

Constant Watchfulness. A trained nurse constantly in attendance is just as necessary in pneumonia as it is in typhoid fever. A sudden delirium or a rapidly occurring cardiac failure may not be observed if no one is in attendance who knows how to interpret symptoms.

The physician should either see the patient twice a day or should be in position to hear from a trained nurse frequent reports of progress during the day and night.

TREATMENT OF SPECIAL SYMPTOMS.

Fever. The temperature in croupous pneumonia ordinarily ranges between 103° and 105° F. (39.4° and 40.5° C.). For eight or nine days this degree of temperature is not par-

ticularly harmful. Baths and sponges are not necessary, and it would seem that the high temperature for that length of time is no more harmful than the disturbance which their administration entails. Cold may be applied to the head or to the affected side of the chest so as to control the temperature to a certain extent. Coal-tar antipyretics should never be used.

Delirium. The delirium of pneumonia can be relieved by methods designed to combat the toxemia, to wit, abundance of fresh air and an abundance of liquid. Bromid of potassium in doses of 20 grains (1.3 Gms.) every two or three hours is of great value. When the delirium occurs in a patient addicted to the use of alcohol, I am quite sure that the use of alcohol in moderately large doses will quiet the patient as will no other drug.

When the patient is violently delirious, thrashing about the bed without sleep, wearing himself out, the use of morphine hypodermically, either alone or combined with hyoscin, is of the greatest amount of value. The patient should not be restrained by means of a strait-jacket or straps, except under dire necessity. The writer has seen patients so exhausted when having to fight against the straps, that exhaustion leading to death has seemed to be the result of the uncontrolled restlessness. Better by great odds the quieting effect of moderate doses of opium than the forcible restraint of the strait-jacket.

Circulatory Disturbances. The circulation in pneumonia is affected apparently in several ways. First, by the bacteremia, in which the patient is poisoned by the bacterial toxin circulating in the blood. In this condition the heart is rapid and feeble, the first sound is weak, and the blood-pressure is frequently low. In regard to the height of the blood-pressure and the prognosis of the case, a low pressure is significant of a severe toxic state. Although occurring just at the height of the sepsis and when death is imminent, there is rising blood-pressure.

The circulation is also affected when there is extensive pulmonary infiltration, when the pulmonary circulation is embarrassed, and when there is much cyanosis. In these conditions there is frequently high blood-pressure.

In the cases with sudden circulatory failure, with decided cyanosis, leaking skin, and running, feeble pulse, there is apparently failure of the vasomotor system, and the patient is virtually in a state of shock.

Each of the conditions of the circulation needs a different treatment.

In the first, with high and often irregular fever, possibly with a low leucocytic count and low muttering delirium, fresh air, cold sponges, normal salt solution either by hypodermoclysis or intravenous injection, or protoclysis, are valuable. Caffein in the form of caffein sodium benzoate, used hypodermically, and strychnin are valuable. Digitalis should be used, but its value is much more problematic.

In the second condition, with dilatation of the right side of the heart and a high blood-pressure, cyanosis and accentuation of the second pulmonic sound, blood-letting is of the greatest amount of value. Here, the use of digitalis is indicated. Other cardiac stimulants, such as caffein and ammonia, also can be called into use here.

The third condition, with sudden fall of blood-prssure, running pulse and leaking skin is the most hopeless of all, and here strychnin, caffein, the intravenous injection of adrenalin chlorid may be tried. In the author's experience, however, all such measures are of little real value. The pressure will rapidly rise, but the hypertension is of extremely short duration. Strophanthin and digitalis must be used hypodermically with the hope of strengthening the failing heart-muscle. Atropin in full doses should be used to overcome the œdema of the lungs.

Oxygen may be used, and will give some comfort to the patient, but there is no lasting value to be obtained thereby.

The Toxemia of Pneumonia. Low, muttering delirium, rapid pulse and leucopenia are a severe and annoying set of symptoms calling for prompt and vigorous treatment.

Here the patient must certainly be given all the fresh air possible. Windows must be opened whatever the degree of temperature of the outside air. The circulation must be supported by strychnin and caffein. An abundance of water by mouth, by rectum, under the skin, or directly into the veins is most helpful. It is just this phase of pneumonia in

which alcohol in some form has been so widely used, and is still a commonly used drug. The author has used both large and small doses of alcohol, in the form of whisky and brandy under these conditions, and after observation he is of the opinion that a case of severe pneumonia is best treated *without the use of large doses of alcohol.*

In a recent series of 25 cases at St. Timothy's Hospital, Roxborough, with a mortality of 12 per cent., all alcohol was withheld. Many of the cases were of a most severe type. I recognize that the number of these cases is small, too small to prove a rule, but their behavior was better than that of an earlier group of pneumonias to which large doses of alcohol were given.

Small doses, ½ ounce (15 mils) every three hours, 4 ounces (120 mils) in twenty-four hours, are perhaps of value because that much alcohol can be used as a food. It is apparently burned up without any trouble, and, therefore, is of value.

Abdominal Distention. This symptom is probably the result of the toxemia, and it is best combated by the methods used to overcome that symptom. In addition, salicylate of eserin, used hypodermically, is of value. Pituitrin will also act well under such circumstances. Turpentine stupes applied hot to the abdomen will sometimes give relief. A rectal tube may also be kept constantly in place during the periods of greatest distention. In using the rectal tube, care must be taken to protect the bed, because frequently thin fecal matter will come through the tube, making an annoying condition.

Acute Dilatation of the Stomach. This condition is more frequently a complication of pneumonia than is supposed. It occurs usually just after the crisis, or after the patient seems well on his way to recovery, following lysis. The patient is usually obstinately constipated, shocked, and vomits large amounts of ill-smelling material from the stomach. There is great distention of the abdomen, especially in the epigastrium, a fullness due to dilatation of the stomach with liquid and gas, and such a clinical picture is frequently mistaken for obstruction of the intestines, but having once been seen and recognized, it is easily diagnosed thereafter.

For this accident the treatment is lavage. Even when there is some doubt as to the diagnosis, the passage of a stomach-tube is demanded. The tube is easily swallowed, and apparently does not greatly disturb the patient, though he seems and may be critically ill. On the contrary, if the diagnosis is correct, the relief is almost instantaneous, the pulse becomes better, the cyanosis and shock less, and in a short time the patient is on the high road to recovery. If the diagnosis is not confirmed by the use of the tube, the patient is not in the least harmed by its passage, and, on the contrary, is somewhat relieved. The dilated stomach needs repeated lavage, frequently about every twelve hours for two or three days. The frequency with which the washing is repeated, and the time over which it must be continued, depend upon the condition of the patient. If the symptoms are entirely relieved, the vomiting does not return, the collapse and constipation are overcome, there is no reason for repetition of the washing of the stomach, but the operation must be continued so long as the symptoms continue.

After the lavage the patient must be placed either upon the right side or upon the face. This relieves the constriction of the intestine which always occurs in these cases, whatever the sequence of dilatation of the stomach, and constriction under the mesenteric notch. The relief given removes the true obstruction which occurs at this spot.

In addition to the measures just detailed, the hypodermic administration of eserin hydrochlorate or sulphate in $\frac{1}{24}$ grain (0.00270 Gm.) doses is of great benefit in acute gastric dilatation, and as a precautionary step the fluid intake of the patient must be decidedly curtailed.

Nephritis. Every severe case of pneumonia shows the presence of a varying amount of albumin and tube-casts in the urine. This is not of moment. However, there is a certain number of cases in which the urine is much diminished and is loaded with tube-casts and albumin. *Do not give stimulating diuretics in these cases.* Give water, limit the nourishment to milk, cup the loins, and use heat over the lumbar region.

Pleurisy. There is, perhaps, no case of croupous pneumonia which is not accompanied by a greater or less inflam-

mation of the pleura. This is manifested by the "stitch in the side," pain on deep inspiration in the adult, and by the expiratory grunt of the afflicted child. A friction rub can usually be demonstrated by auscultation.

The treatment of pleuritis is strapping the chest, just as would be done in a broken rib, and the application of cold in the form of an ice-bag or an ice-coil. The use of either dry or wet cups gives a great amount of relief. If the pain is severe, the administration of opium, usually as morphin, hypodermically, is absolutely necessary.

This simple pleurisy is the rule in pneumonia, but occasionally it is accompanied by a serous effusion. The treatment of this simple serous effusion, which occurs during the height of the disease, depends upon the mechanical effect of the effusion. If the heart is not much displaced, if there is little embarrassment of breathing, then the effusion may be safely left to the forces of nature for its absorption. If, on the other hand, the effusion embarrasses the patient in any way, the fluid should be aspirated under the strictest aseptic precautions.

If this tapping of the chest is done under local anesthesia, and the skin incised so that the needle will easily enter the chest, there will be scarcely any disturbance of the patient. On the other hand, if the needle be plunged through the skin without first using a local anesthesia and incising the skin, great shock may be produced. The writer has seen death occur as the result of such a shock.

Purulent Pleurisy. When the temperature of a case of pneumonia begins to rise after the crisis or lysis, or when the temperature does not fall after eight or ten days, especially if it takes on the character of an increased septic fever, and the leucocytes increase in number, the physician knows that the patient is the subject of some active inflammatory complication. The most common complication of this kind is empyema. The fluid may be either free in the pleural cavity or confined between the lobes, or between the lower lobe and the diaphragm.

Frequently repeated careful examinations of the chest are necessary to locate the liquid. If it is free in the pleural cavity the diagnosis is easy, the only necessity being daily

observation of the naked chest. There is flatness over the chest, decreased tactile fremitus, and loss of voice sounds. The heart is pushed to the right or left, the liver displaced downward if the effusion is on the right side, and the area of gastric tympany diminished if the fluid is on the left side. In children the physical signs are often misleading. A puncture with a sterile aspirating needle will establish the diagnosis. The diagnosis, however, is not so clear-cut and easy if the liquid is confined between the lobes. In this condition the flatness is frequently localized in the line of the interlobar fissure. There is lack of fremitus, with decreased voice and breath sounds over this area, sometimes accompanied by a circumscribed edema of the skin. If there is doubt as to the presence of liquid, a needle should be inserted between the ribs and careful search made with the point plunged in different directions. Unfortunately for this method of diagnosis, the needle often does not reach the liquid, even although the physical signs are positive. The difficulty is that either the needle is too small or too short or has not reached the collection of pus. If the patient is near an *x*-ray apparatus, a good stereoscopic picture should be taken in order to prove the presence or absence of a purulent focus. Not only does the *x*-ray show the shadow of the pus, but it shows its exact position. The presence of a collection of pus being once established, the fluid must be evacuated.

This operation occasionally is the simplest of procedures, and may be done by any physician who is at all accustomed to the technic involved. But it is by all odds the safest plan to send such a patient to a hospital and have the chest carefully exposed under anesthesia. One word of caution: When the patient is a child and the chest is full of liquid under much tension, the sudden withdrawal of the liquid by means of an incision is often followed by a severe shock, sometimes even by fatal collapse. Therefore, it is a safe rule to tap first, and afterward to drain surgically.

Arthritis. Certain cases of pneumonia are followed by arthritis, or the inflammation of the joint may occur during the height of the disease. It must be remembered that these attacks of pain and swelling of the joints signify a localization of the toxin, or, perhaps, of the pneumococci themselves.

The treatment is fixation of the joint and the application of a cool evaporating liquid, such as lead-water and laudanum, or saturated solution of magnesium sulphate. Occasionally the joint becomes purulent, and in this event the surgeon's aid is to be enlisted.

Middle-ear Disease. This condition often appears insidiously, particularly if the patient be very ill. The ears should be regularly examined, and particularly if there is an unaccountable rise of temperature or a continuance of temperature after it should normally fall. If the ear-drum bulges, it should at once be punctured.

Meningitis. This is an inflammation of the meninges due to the presence of pneumococci in the cerebrospinal axis. It is manifested by the ordinary symptoms of meningitis, stiffness of the neck and back, delirium and Kernig's sign. It may be confused with the so-called meningismus, due to the irritation of the meninges by the pneumotoxin. The diagnosis is made by spinal puncture, and the best treatment is this same puncture repeated every twenty-four hours, or even oftener if symptoms demand it.

Treatment of the Crisis. When the crisis occurs the patient is getting well. There is no danger of collapse on account of the suddenly lowered temperature. On the contrary, the pulse drops in frequency, and the heart becomes slower and more forcible. The patient expresses himself as feeling well. Therefore, the only treatment is to see that because he feels so well he does not abuse his condition by attempting to sit up, by much talking, or by other undue exertion. Stimulants are rarely ever needed.

THE USE OF EFFICIENT DRUGS.

Digitalis. This, in the writer's experience, is the most useful single drug in all cases of pneumonia with an organic heart disease. Digitalis should be given from the beginning of the attack in moderate doses, 10 drops (0.6 mil) of a good tincture three times in the twenty-four hours. This dose can be increased if the heart shows signs of dilatation; 10 drops (0.6 mil) used by the mouth or hypodermically can be given every three hours, care being taken that the quantity given is not large enough to cause poisoning by this most useful

drug. Remember that when one uses digitalis one does so because the patient surely needs help. Therefore, see that the digitalis is obtained from a source which will insure a potent article. Good digitalis may be life-saving, while a worthless drug may sacrifice a life that otherwise could be saved.

Strychnin. Pharmacologists tell us that strychnin neither raises the blood-pressure nor stimulates the heart. Be that as it may, doses of $\frac{1}{30}$ grain (0.00216 Gm.) every three or four hours, used hypodermically, are of great value. The patient rests better, his pulse is stronger, and he is distinctly improved by its use. Therefore, in severe cases where there is toxemia, and where the circulation is poor, I believe that full doses of strychnin are not only indicated, but are useful.

Morphin, in violent delirium, when the patient is restless from his toxemia, when he has pain from his pleurisy, is of the greatest value. It should be used with caution, however, care being taken to avoid narcosis.

Atropin. This drug, used when there is a tendency to edema of the lungs, is of much value. It should be used in doses of $\frac{1}{100}$ of a grain (0.00065 Gm.), repeated every two or three hours if necessary.

Caffein. The alkaloid in 2- or 3- grain (0.13 or 0.195 Gm.) doses by the mouth, or caffein sodium benzoate hypodermically, is of value when the patient is weak, either from grave toxemia or from failure of the circulation.

Camphor. In the form of camphorated oil used hypodermically, 1 or 3 grains (0.065 or 0.195 Gms.) of camphor every one or two hours is useful. It is by no means a specific, but sometimes will help to lift the patient over the incline.

Nitroglycerin. This is a much abused drug. It is *not* a heart stimulant. The pulse becomes fuller after its use, but this is because the peripheral vessels are dilated. It is of apparent use when the heart is laboring and fighting against a high pressure. Then the peripheral vessels are enough dilated by the use of the drug to allow the heart to become more efficient. Cyanosis, with a laboring right heart, are the indications for its use. It should be used in full doses for a short time. It should not be used in severe toxemia,

Oxygen. This gives relief to the patient with much cyanosis and laboring heart. The respirations are quieted, and the heart labors less. It is a good adjunct to fresh air. Fresh air, however, will usually make its use unnecessary.

Calomel. If there is much abdominal distention, this will do good by causing free bowel movements, and by apparently lessening the tendency to fermentation in the intestinal tract.

Eserin and Pituitary Extract. These are of value where there is much abdominal distention. They appear to cause the expulsion of gas from the intestinal tract by stimulating the muscular coat of the gut.

CATARRHAL PNEUMONIA.

Catarrhal pneumonia is a bacterial disease affecting the lobules of the lung. It differs from croupous pneumonia in several respects. The various groups of bronchopneumonia have a different beginning from those of croupous pneumonia. While croupous pneumonia begins suddenly, with chills, high fever, and rapid respiration, only in exceptional cases of bronchopneumonia does this sequence occur.

Certain cases, particularly in children, and occasionally in adults, begin acutely, just as cases of croupous pneumonia do. These cases, however, are much rarer than are the ordinary ones of bronchopneumonia which follow fevers, such as measles, whooping-cough, influenza, scarlet fever, and so forth. The bacteriology of this disease also differs from that of croupous pneumonia. In croupous pneumonia, the largest number of cases are due to one or the other types of pneumococcus, whereas, in catarrhal pneumonia, the influenza bacillus, the micrococcus catarrhalis, the pneumococcus, Friedländer's bacillus and various streptococci are very much more frequently the etiologic factors.

Three divisions of this bronchopneumonia are usually recognized—the acute type, which resembles very closely in its symptoms and course croupous pneumonia; the form which follows infectious fevers, such as measles, scarlet fever, diphtheria, influenza and whooping-cough; and the so-called aspiration pneumonia, which is likely to follow certain depressing states, in cases of etherization, and particularly in older individuals with nose and throat conditions.

The symptomatology of the acute cases is very much the same as that of croupous pneumonia, and the course is very much the same. The ordinary bronchopneumonia which follows measles, whooping-cough, influenza, inflammation of the lungs, is limited to smaller areas. The inflammation begins usually in the smaller bronchi, and in the lobules of the lung. The primary condition, for instance, may be progressing normally, and even begin convalescence, when the temperature rises, the course of the disease becomes prolonged, the child begins to cough more frequently, a leucocytosis occurs, and one realizes that one is in the presence of some complication. Coincident with this increase of symptoms, and particularly the pulmonary symptoms, physical signs may make their appearance in the lung. Here there are small areas of dullness, scattered in various portions of the lungs, larger or smaller, depending upon the severity of the disease and the portion of the lung affected.

If these areas are isolated and large enough, blowing breathing, together with increased fremitus, and increased vocal resonance may be part of the physical signs. The pulse becomes rapid, the respiration more frequent, and the child cyanosed. With the progress of the disease the cyanosis increases, urgent dyspnea occurs, and the child may die of respiratory failure, due to involvement of large areas of the lung.

The disease is often prolonged for several weeks, far beyond the time at which a croupous pneumonia begins to resolve. Then the question, as to whether the subject has tuberculosis or not becomes a very grave one. In the majority of cases, this can be settled only by the termination of the case into complete health, or by a frank tuberculous process in the lung.

As a preventive measure of bronchopneumonia in a child sick with an exhausting disease like measles, diphtheria, or influenza, the room in which they are nursed should be well ventilated, and the child carefully protected from draft. The chilling of the body apparently, has much to do with the onset of the pneumonia in certain areas of the lung. If the child is protected, however, the temperature of the room does not need to be of a uniform character.

TREATMENT.

The general principles of treatment differ little, if at all, from those applied to the treatment of croupous pneumonia. The patient should be kept in bed in the fresh air, the windows of the room being wide open. The patient should be thoroughly protected from chilling by the use of proper bed clothing, and proper underclothing, such as combination drawers if he is a child, and if the weather is very cold, a cap should be placed on the head, and mittens on the hands. The drawers should be made with stocking feet, so that in case the child becomes uncovered, the body will still be protected by a set of drawers.

The child should be given an abundance of water, simply as a method of lessening the intoxication, and he should be fed milk and soft foods such as toast, eggs, junket, and mashed potatoes. The use of alcohol in this condition, follows the same rules as the use of alcohol in other infections—it should be given in amounts large enough to act as a food. For a child 5 years old a half an ounce (15 mils) in twenty-four hours is an abundance. Larger doses depress rather than stimulate.

As the disease is likely to be prolonged, particularly in the secondary cases, the fever, which may last two or three weeks, reaching 102° or 103° F. (38.9° or 39.2° C.), should be adequately controlled, the best method being by sponging, or by tubbing. The temperature of these sponges and baths should be relatively high, from 90° to 100° F. (32.2° to 37.8° C.). Coal tar preparations should never be used, inasmuch as they depress the heart, and do more harm than good.

Applications to the chest are of some value. When the child is much depressed by a great degree of diffuse bronchitis, as evidenced by rapid breathing and many râles over the entire chest, applications of mustard water to the chest, afterward surrounding the chest with a padded cotton jacket, often causes such local irritation in the skin, that the congestion of the lung is apparently relieved. The old-fashioned flaxseed poultice entirely surrounding the chest is of value in advanced cases. More valuable than these flaxseed poultices, because more easily applied, is the quilted cotton jacket surrounded by oiled silk or oiled muslin. This is closely applied to the child's

chest, and makes a veritable poultice in a very short time. This jacket should be frequently changed for cleanliness sake, and while it is being changed, the skin should be rubbed thoroughly with alcohol or with a weak mustard water. When the child's breathing is much oppressed, when he is cyanosed, when the same fine subcrepitant râles are heard over the entire chest, intermittent dipping in warm and cold water will occasionally bring about so much relief, that the child may actually be turned toward the road to recovery, instead of going down hill as he apparently was about to do. A bath about a temperature of 110° F. (43.3° C.), and another one of a temperature of about 85° or 90° F. (28.9° or 32.2° C.) is useful. The child is first stripped, dipped into the tepid bath, and then quickly dipped into the colder bath, then into the tepid and again into the cold. In this way deep inspirations are forced, and the exudate is cleared from the small bronchi; the child again breathes easily. I am quite sure that I have seen life saved by this method of treatment, when the condition was as stated.

Internal medicines are of value. Just how to use them, and just how much to use, however, is a question depending upon each individual patient. Two drugs it seems to me are of paramount importance: chlorid of ammonium, and carbonate of ammonium, in cases where there is a sticky, viscid exudate, and the chest is full of fine râles. These drugs given in proper doses, well diluted, relieve this condition very markedly. A child three and one-half years old should get 1½ to 2 grains (0.090 to 0.120 Gm.) of chlorid of ammonium, or 1 grain (0.065 Gm.) of carbonate of ammonium, repeated every two or three hours according to the condition of the case. Citrate of potassium, under certain conditions, in 3-grain (0.195 Gm.) doses is of value. It softens up the mucus and viscid exudate, and relieves the case in this way. Where the bronchitis rapidly increases, and spreads over both lungs, the use of atropin in the form of tincture of belladonna, is often of the greatest value. It dries up the mucus, which often is not the result of an actual inflammation, but represents rather of an exudate into the bronchi, and in that way clears the bronchial tubes.

When the heart is rapid and dilated, the use of digitalis is of the greatest importance. I am quite sure that I have seen

cases recover by doubling the dose of digitalis and belladonna, which were being steadily given. This of course can be judged only by the dilatation of the heart, and by the amount of obstruction to the breathing.

Strychnin, in a dose of $\frac{1}{120}$ of a grain (0.00365 Gm.) every three hours, in a child three or four years, is a dependable drug, but care must be taken not to give enough to cause poisonous symptoms.

When a child coughs constantly, frequent stimulation of the chest wall by mustard baths, or by a poultice containing small amounts of mustard, very often relieves the cough. Great care should be used in the administration of opiates under these conditions, and the milder opiates should always be chosen. The best one is paregoric. The dose of this is so easily regulated, that there is little danger of giving an overdose, as there would be if morphin, or one of the other alkaloids was used. Ten to fifteen drops (0.8 to 1 mil) of paregoric to a child two years old, given every three hours, will be sufficient, for the first dose. If this does not control the cough and if there is no sign of narcosis, then begin gradually to increase the dose two or three drops at each dose, until some effect is produced. But I think the use of opiates is generally contraindicated in children with this condition. The opium seems to lessen the response of the respiratory centers to stimulation, and makes the patient less able to expectorate.

When the cyanosis is extreme, the use of oxygen is of value, but there is no oxygen quite so good as the use of pure, circulating, fresh air, with the child thoroughly protected against chilling of the surface of the body.

PYOGENIC INFECTIONS.

(Septicemia, Pyemia, Toxemia.)

This infection has its origin in any lesion of the body, be it superficial or deep, which becomes infected with any of the pyogenic bacteria. A mere scratch on the skin, if a virulent streptococcus becomes implanted and grows, may give rise to the most violent fever, chills, exhaustion, and even death.

The streptococcus is not the only invading micro-organism, however, for any pathogenic bacteria, such as staphylo-

cocci, gonococci, colon bacilli and pneumococci may give rise to the most violent symptoms, the streptococcus and the staphylococcus being the most frequent invading micro-organisms. Puerperal fever is simply an infection in which the seat of the disease is the female genital tract, the germs getting their lodgment from uncleanly nurses or obstetricians, although occasionally some local lesion present before labor is the primary cause.

Septicemia is the name given to the condition where there is an infection of the blood-stream arising from some primary focus, and where there are no secondary abscesses consequent to the bacteremia.

Pyemia always results from a septicemia, and the name pyemia is applied to a condition of multiple foci of infection resulting from the original septic infection. Septic endocarditis is but a phase of pyemia.

The term toxemia is applied to the condition in which the symptoms are due to toxins which result from the multiplication or breaking down of the bacteria, or destruction of the cellular elements of the body.

The mortality of septicemia and pyemia resulting from the streptococcus and staphylococcus is extremely high, according to Pearce and Austin—83 to 88 per cent. If the multiple abscesses form in the internal organs such as the liver, kidney, spleen and brain, pyemia almost always has a fatal ending.

TREATMENT.

Prophylaxis is perhaps the most important part of the treatment, and to insure this prevention of pyemia prompt surgical treatment of superficial wounds is imperative. Asepsis in the conduct of surgical and obstetrical operations are of the first importance as a prophylactic safeguard.

A mere scratch may give rise to fatal consequences by the time the original wound is brought to the attention of the general practitioner. Such a wound should be treated thoroughly, being opened far beyond the limits of the original abrasion, and thoroughly sterilized with antiseptic solutions, and, if necessary, with iodin.

Fear of giving pain should not interfere with the free use of a sterile scalpel. This is especially important where the

wound has been contaminated by street dirt and a potential tetanus infection may be implanted.

No physician should undertake the simplest surgical operation, to say nothing of major operations, unless he uses every precaution to prevent infection of the otherwise clean wound. No physician should attempt to conduct the simplest labor case unless he recognizes that his handling of the genital tract of the patient may give rise to septicemia, and unless he views the case as one of major surgical importance.

After infection has taken place, the original seat of invasion must be thoroughly exposed, drained, and packed, if such procedure is in keeping with the safety of the patient. If the origin of the infection is about the teeth, these should be subjected to the most thorough treatment, even to their extraction. If the original site is in the genitourinary tract, this must be flushed by irrigation of the bladder and sometimes of the pelvis of the kidney.

General treatment must at once be instituted.

Free use of fresh air, sunlight, and the drinking of large amounts of water are the best means. The temperature must be controlled by the circulation of fresh air, and the application of cold to the surface of the body by means of cold sponging or cold baths is to be made.

Coal-tar preparations must never be used. They will lower the temperature, but they do harm. Water may be used by the mouth, by enteroclysis, and by intravenous injections.

Alcohol in small quantities may be given, but it is harmful if given in intoxicating doses.

Food is of the highest importance. Large quantities of milk should be used.

Strychnin, in doses of $\frac{1}{30}$ grain (0.002 Gm.), every three hours, is useful.

Digitalis may be used either hypodermically or by the mouth.

Lately the use of foreign porteids in the form of killed bacilli of almost any type have been used with apparent good effect in certain localized infections, especially in the cases of uncontrolled arthritis. Given in small quantities into the vein, it is followed by a severe reaction, often by a chill and

high fever. After the subsidence of the reaction there is a remarkable amelioration, but unless the cause is removed the conditions return.

In certain local infections the use of autogenous vaccines has proven of signal value. However, the type of the micro-organism must first be ascertained, if any hope of a specific cure is to be realized. Without a knowledge of the character of the infecting germ, and without the certainty that the bacteria in the vaccine and the infecting focus are of the same character, vaccine treatment is a slipshod, uncertain method of therapeutics, and should not be used.

Vaccine therapy, to be of value, must be exact, and any other method of its use is highly irrational.

In diphtheria, tetanus, epidemic meningitis, and possibly in influenza and cholera, the sera are specific and life-saving. In streptococcemia they have not been proved of value because of the weakness of the sera, and because of the uncertainty that the sera are made of the same strain as the infecting organism.

COLON-BACILLUS INFECTIONS.

The colon bacillus is a normal inhabitant of the healthy human intestine, but under certain conditions the micro-organism becomes pathogenic, and may give rise to a local infection or to a general disease. Infections of the bladder and kidney, certain forms of arthritis and synovitis are familiar examples of the local colon infection. Other cases give rise to a general infection characterized by rather long-continued, inexplicable fever.

The diagnosis of a colon infection can be made only by agglutination reactions, and by recovery of the bacillus from the blood of the patient, or from the seat of the local infection.

In all cases of arthritis, cystitis, pyelitis, cirrhosis of the liver, severe anemia and gall-bladder disease, and in all cases of fever entirely inexplicable in type, it must be remembered that the colon bacillus may be the bacterium responsible for the clinical picture of the patient under consideration.

The treatment of a general colon infection, which often closely resembles typhoid fever in its symptoms and course,

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must be based upon the general lines of treatment of that disease.

The use of an autogenous vaccine may possibly serve as a certain antidote.

Local infections, such as cystitis and pyelitis, may be diagnosed by examination under proper precautions of the bladder and renal pelvis, and by the study of cultures made from the exudate from these parts. If they are found to be due to a colon bacillus, autogenous vaccines may be used with a certain expectation of success. Large amounts of water and freedom from irritating food will be of value.

If the case is one of a chronic disorder, such as cirrhosis of the liver, anemia or changes in the spinal cord, evacuation of the intestinal tract, the daily use of high colon irrigations and the administration of mineral oil may be found of benefit in controlling the infection. While there are certain well-defined and positive cases of colon infection, the subject lends itself to much misinterpretation. The laity, always naturally desirous for the knowledge of the name of his ailment and for its cause, will readily grasp at "colon infection," whether the title is justified or not. Hence, the conscientious physician will be as certain as possible of his data before he announces the name of the illness to the patient.

DIPHTHERIA.*

Diphtheria is an acute contagious disease caused by the Klebs-Löffler bacillus. It is characterized, clinically, by the formation of a membrane almost always upon the upper respiratory tract (pharynx, larynx and nose), and by secondary systemic changes resulting from bacterial toxins. Other portions of the body, such as mucous and serous membranes and wounds, may harbor the bacteria locally, and it has been stated that a diphtheritic toxemia may exist without the actual presence of a membrane. These are rare, and unusual manifestations of the disease. In common parlance, diphtheria refers to an angina caused by the Klebs-Löffler bacillus with the resultant diphtheritic toxemia.

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The Klebs-Löffler bacillus, first described in 1883, varies greatly in different cultures and strains, and expert knowledge is often required to distinguish some of the more unusual forms. It is doubtful whether diphtheria, as a clinical entity, should ever be diagnosed by culture alone in the absence of the general clinical signs of the disease.

Diphtheria is endemic in all the large communities in America and most parts of Europe. At times it may become epidemic, and this may occur also in smaller communities and institutions. It is more prevalent in winter than in summer. It affects all ages and both sexes. It is largely, however, a disease of children under 10 years of age, although its occurrence in adults and older children is by no means rare. Very young children may be affected by the disease, even infants but a few months of age. It is about equally divided between the sexes, although some observers have stated it to be more prevalent amongst girls.

Diphtheria is transmitted from one patient to another by means of the buccal, pharyngeal and nasal discharges. It may be transmitted directly, as is more often the case, or indirectly by means of intermediate carriers, animate or inanimate. The disease must be considered communicable as long as virulent diphtheria bacilli exist at the site of a given lesion. Certain outbreaks of diphtheria have been traced apparently to infected milk. Mild unrecognized cases of diphtheria are perhaps amongst the most important factors in the continued prevalence of a disease so successfully treated when recognized.

The local pathologic lesion produced by the diphtheria bacillus is the formation of a membrane, consequent to the process of coagulation necrosis. This membrane finds its most frequent seat upon the tonsils and fauces, and by extension spreads to the nose, larynx, and even to the trachea and bronchi. The inflammation of the tonsils and pharynx is, in general, deeper and more severe than that of the larynx or bronchi. Implication of the mouth is rare. The other local complication is otitis, which may be complicated by mastoiditis. The latter condition, however, is exceedingly rare. Cervical adenitis is not uncommon.

The lesions due to the diphtheria toxins are many and important. Renal complication, either as an albuminuria or

as a true nephritis, is common. The liver and spleen are often involved. Myocarditis is common. The most characteristic general lesion of diphtheria is an inflammation of the nerve roots and sheaths, leading to palsies of various kinds. Pneumogastric degeneration is not uncommon.

The symptoms of diphtheria vary with the severity of the disease, and the locality and extent of the lesions. Briefly stated, the cardinal early symptoms are sore throat accompanied by fever, which, however, is not usually of very high range. The pulse is comparatively rapid, and after the onset of the disease, is not as strong as in many other acute febrile conditions of the same temperature range.

The diagnosis is made by the appearance of the characteristic membrane. Usually appearing first upon the tonsils and spreading therefrom, it shows itself as a whitish-gray membrane or film. If seen before this membrane appears, the throat shows an erythema, and may be positive to culture. The membrane, beside extending, may change in color somewhat. In any event, it is usually quite adherent. In cases untreated by antitoxin the membrane begins to free itself within five to seven days. When antitoxin is used in adequate dosage, the progress of the disease is arrested, and separation of the membrane begins to take place within a few hours.

Constitutional symptoms—that is, those of toxemia and cardiac weakness—are noted early, unless the case be of the very mildest type.

The most frequent clinical complications are cardiac failure, paralyses, and broncho-pneumonia in the laryngeal types.

The diagnosis rests upon a double base. Besides the clinical signs mentioned, we have an aid in the making of smears and cultures from the infected areas. These are of the greatest assistance when correctly made, but in the hands of the inexperienced often lead to error.

The differential diagnosis of diphtheria offers but one serious difficulty. In certain cases of scarlet fever the angina resembles that of diphtheria to such an extent that differentiation must be made by repeated culture or by the accompanying clinical picture of scarlet fever. It must not be forgotten that scarlet fever and diphtheria not infrequently co-exist. Various forms of tonsillitis, peritonsillar abscess,

and Vincent's angina should offer but slight difficulty in differentiation from diphtheria.

The resemblance of the laryngitis of incipient measles to diphtheritic croup or laryngeal diphtheria is often so marked as to make an immediate and certain diagnosis impossible.

In cases in which a reasonable suspicion of diphtheria exists the matter of diagnosis will be simplified by the use of antitoxin which can never do harm.

TREATMENT.

In the treatment of diphtheria we are infinitely better situated than in dealing with other contagious diseases, because we have at our disposal a definite physiologic remedial agent. Following the researches of Behring, an antitoxin for diphtheria has been perfected, which in practice consists of the serum of the horse immunized by the injection, in progressively increasing doses, of diphtheria toxins. For convenience in description and administration, a unit of diphtheria antitoxin has been established. A unit is that quantity of antitoxin "that will just neutralize 100 minimal fatal doses of toxin for a 250-gram guinea-pig."

The action of the antitoxin may be described as tending to the production of passive immunity. It does not primarily have any other action or expected effect than of itself to neutralize the toxins produced by the diphtheria bacillus within the body of the patient either before or after the use of the antitoxin, and to aid in destroying the bacilli themselves.

The antitoxin is administered either subcutaneously, intramuscularly or intravenously. While to the experienced operator intravenous injection is easy, it is a procedure of sufficient difficulty to render it undesirable as a routine measure in the hands of the occasional user.

I prefer the intramuscular injection, believing that it causes least discomfort, and that a dose thus given has a prompter and more efficacious action than when an equal or larger dose is given subcutaneously.

The syringe and needle having been sterilized, the antitoxin is drawn into the syringe with care. Where the antitoxin supplied commercially is used it is almost always within

the syringe as a first container. The point of election for the injection is the anterior external surface of the thigh. The area for the introduction of the needle is prepared by the application of tincture of iodin. The needle is thrust well into the muscle, and if the injection be not made with too great rapidity great pain is not caused. After the withdrawal of the needle the opening is closed by the use of a small pledget of cotton and collodion.

The dosage of antitoxin is still a matter of opinion, and to a certain extent must always remain so. It must be determined by:

1. The location and severity of the local diphtheritic process.
2. The patient's general condition.
3. The patient's age.
4. The day of the disease upon which it is given.

There are certain factors, in our estimation, of the dosage which render it impossible to fix a mathematically correct dose. Most important of these is the fact that we cannot accurately determine the virulence of a particular attack; in other words, express definitely the amount of toxemia we have to deal with. The dosage, therefore, must always be based upon experience, and be, to a certain extent, empirical.

The day of the disease is of importance also; it is evident that the sooner the antitoxin is given the less will be needed. It is a matter of common experience that antitoxin given after the third day is much less efficacious, and must be given in much greater doses. We must bend all our endeavors to the prompt administration of antitoxin.

The age of the patient is not of the greatest importance, except in infants. Children under the age of 2 are given about one-half the dose required for other patients, and we rarely give less than 5000 units, except to infants.

A severe infection requires a larger dose than a mild one, except that we do not ever reduce our dosage below a certain working minimum sufficient to meet the probable requirements in a given case.

The antitoxin is to be given promptly, and in sufficient dosage to abate the disease quickly, thus ensuring recovery and lessening the incidence of complications or sequelæ. It

is, therefore, desirable to give sufficient antitoxin *at the first injection* to meet the needs of the patient. Generally, if the antitoxin be administered early this can be accomplished. But if, within twelve to eighteen hours, the membrane appears to be spreading, a second dose should be given.

The beneficial action of the antitoxin manifests itself clearly. It is evidenced by the improvement in the patient's general condition, by lessening of the evidences of toxemia, and by a prompt improvement in the local lesions of the disease. The membrane, wherever situated, within a few hours shows, first, a cessation of its spread, if this has still been active; and, secondly, marked evidences of separation of the membrane already present, and soon, signs of healing beneath.

As has been stated, the dosage of diphtheria antitoxin must be such that a very prompt improvement is noted in almost every instance.

In general terms, it may be formulated thus:

In tonsillar cases, those in which the membrane is strictly limited here, the dosage on the first day is 5000 units. On the second day the initial dosage would be 10,000 to 20,000 units, while after the second day from 20,000 to 40,000 units, and even more would be given as an initial dose. In faucial diphtheria about the same dosage holds good.

In laryngeal diphtheria, when there is no dyspnea, the initial dosage, if the case is seen on the first day, should be 10,000 units, or if there is the least sign of dyspnea, 15,000 units. If seen first upon the second day such a case should receive at least from 15,000 to 20,000 units, and if seen later than the second day 20,000 or more units should be given.

Nasal cases are really of two distinct varieties, and this influences our treatment also. Where there is but slight moisture from the nares, and the diagnosis is made by culture only, the initial dose, if the case is seen on the first day, should be 10,000 units, the dosage again increasing as the case is seen later in the disease.

If, however, there is a distinct nasal membrane, with discharge and any sign of toxemia, 20,000 units should be given as an initial dose, if the patient is seen on the first day, this first dosage going as high as 30,000 to 50,000 units if the

patient is given his first dose of antitoxin as late as the third day.

The maximum total dosage that may be given to a patient in the course of the disease is limited more by our difficulty in procuring for a given case as much antitoxin as seems desirable than by any ill effects from large doses. Three hundred thousand units, with recovery, have been given in a single case.

The complications in the use of antitoxin are rarely of moment, and in any case not the result of the antitoxic substances, but rather of the vehicle, the horse serum itself.

Abscess following correct use of sterile serum is rare, although a pressure decomposition has been known to occur where very large quantities of the antitoxin have been used. This occurrence is extremely rare with the intramuscular use of the antitoxin. Pain and tenderness following the injection are usually of no great moment, especially with the intramuscular method of administration.

If an abscess occur, incision and drainage are indicated. Simple transient induration, if annoying, will usually yield to hot fomentations or poultices.

Serum sickness is a term applied to that series of phenomena which, in susceptible individuals, follows the injection of a serum (*i.e.*, a foreign protein). It manifests itself as an urticarial, morbilliform, or scarlatinaform rash, accompanied at times by fever, nausea, vomiting, joint pains, edema of face and joints, and general malaise. In slight forms it is not uncommon after the use of diphtheria antitoxin, even in the smaller doses, many individuals being very susceptible. It requires no special treatment and is self-limited.

Anaphylaxis is a subject of greater importance, although not entitled to that prominence in the discussion of the use of diphtheria antitoxin which has often been given it. It may be defined (Rosenau) as "a condition of unusual or exaggerated susceptibility of the organism to foreign proteins." It is called forth by the administration of a second dose, as of serum, when the patient has been rendered sensitive by a first dose. In its milder forms it resembles serum-sickness. Graver forms, even ending in collapse and death, have been reported, but in human beings it is an occurrence of such

rarity that it may be left entirely out of consideration in the use of antitoxin. The danger is so slight and the antitoxin so necessary that there should be no hesitation in its use when indicated.

The prophylactic use of diphtheria antitoxin is well established, and in it there is a field of great usefulness. The average prophylactic dose is 1500 units. The protection therefrom is, however, not of very great duration, sometimes as short as ten days. The fact that we have at our command a specific antitoxin for diphtheria should not lead us to overlook the importance of the general medical and systemic treatment of the disease.

Since we know the infectious agent, we must, in addition to enforcing the rules for quarantine and isolation, just as described in the treatment of scarlet fever, be especially on our guard to avoid any semblance of carelessness after contact with a patient or with infectious material. It is probable that diphtheria is carried from one patient to another by means that are entirely avoidable.

The general hygienic and medical treatment of diphtheria offers but few difficulties. The room, preferably large, airy and well ventilated, should be kept at a temperature of about 65° F. (18.3° C.). The patient may receive a daily tepid bath if there is no contraindication. This must be given in bed, so that it involves no exertion on the part of the patient. A cardiac derangement of any moment may make it undesirable or dangerous even to move a patient sufficiently to give him a full bath on any one day.

In the acute stage of diphtheria liquid diet is indicated, and this should be continued for three or four days after the local diphtheritic process is well. After this period the diet may be cautiously increased by the addition of stewed fruits, gelatins, puddings, gruels and well-prepared cereals. Full diet may be given after convalescence is well established and the kidneys normal.

Difficulties in feeding diphtheria patients may arise when there is paralysis of the pharyngeal muscles. When this renders swallowing impossible, nasal feeding must be resorted to.

Water should be given freely in diphtheria, not only because of the fever present, but also to minimize the tendency to nephritis.

The bowels must be kept open. In infants castor oil should be given; in older patients salines, citrated magnesia or other laxatives may be substituted. Particularly when there is a cardiac complication must things be so managed that there is no straining at stool, as this may cause severe, and even fatal collapse. Purging should be avoided at all times.

The diphtheria patient should be kept in bed in the mildest of cases for at least three weeks, and from five weeks upward when there is any demonstrable cardiac or nervous involvement.

The management of cases of laryngeal diphtheria presents a problem somewhat different from that of a simple pharyngeal one. In the former we have to treat not only an intoxication, but also a mechanical obstruction to breathing, with possible asphyxiation.

It often seems that by keeping the patient somewhat warmer than in other cases, and by the use of steam by the croup-kettle and cover, if free access of air be allowed the patient, we can accomplish something to make breathing easier.

In the use of steam in laryngeal diphtheria care should be taken not to employ it to the exclusion of the air. A simple canopy, with the spout of the croup-kettle so situated as to allow the steam to flow toward the patient's head, will be sufficient. Oftentimes the patient will show evidence of the relief afforded by placing the head directly in the path of the current of steam.

For the treatment of the dyspnea of laryngeal diphtheria I have always found the application of turpentine stupes to the neck and upper chest to be of decided advantage. This is evidenced by the fact that adults and older children will very often express a feeling of relief as soon as the application has been made. There are several methods of applying stupes. Ol. terebenthinæ, 2 fluidrams (7.5 mils); ol. olivæ, 1 fluidram (3.75 mils), in hot water, 2 pints (1l.), should be prepared in a 2-quart pitcher, and then poured into a basin as needed. A piece of flannel one-half yard square should be wrung out of this solution, fluffed, tested as to heat, and then snugly applied to the larynx and upper chest. This should be

changed every few minutes, the idea being to have the flannel always as warm as comfort will permit. As an emergency measure, the thorough rubbing of the larynx and chest with camphorated oil, and the subsequent application of the hot flannel, will do good. The heating of the flannel on the chimney of an ordinary coal-oil lamp will oftentimes furnish sufficient heat when hot water is not immediately available.

The internal administration of ammonium carbonate and syrup of ipecac is supposed to be of advantage in hastening separation of the membrane. Should these measures be of no avail, then operative interference must be resorted to in the form of intubation or tracheotomy.

The indications for operative interference are well-marked dyspnea, with recession of the soft parts of the chest, restlessness, and a weak, rapid pulse, especially if these symptoms do not show signs of abating after the serum and other treatment have been administered.

The greatest of care should be exercised always in determining when the time for operative interference has arrived. Operation should not be done too early. The manipulation, with its resultant damage to the soft parts, and the presence of the tube, which acts as a foreign body, may add to the dangers of an already dangerous situation, and should be avoided whenever possible. At the same time the too-long delay in interfering, with a too-great loss of strength, may leave the patient unable to do his part, even after an otherwise most successful operation.

Intubation is the operation of choice in institutions, or where trained attendants are close at hand. Tracheotomy is, broadly speaking, to be preferred in general practise.

Intubation. This should be done always with the patient in the recumbent posture. With the hands by the sides, the patient is rolled and pinned securely in a sheet. The head is then thrown back by having the neck and shoulders rest upon a pillow or rolled blanket. An assistant sitting at the head of the bed or table holds the head of the patient, at the same time keeping the mouth-gag in position on the left side of the patient's mouth. The operator then passes very gently his left index finger along the tongue to its base, where the epiglottis is found and turned backward, the finger then entering

the larynx and resting upon its posterior wall, care being taken not to cause obstruction to the intake of air. The operator with his hand then passes the tube back until its point comes in contact with the left index finger. Then, with the finger as a guide, the end of the tube is engaged in the larynx, the handle of the introducer is slightly raised, and the tube allowed to drop in with very little pressure. The tube is then disengaged from the obturator and allowed to remain. The string, or what is much better, a No. 4 banjo wire, securely looped in the hole in the head of the tube, should be allowed to remain in place, and attached on the outside of patient's cheek by means of a piece of adhesive plaster. The string, or wire, should be drawn fairly taut for fear of extubation with the tongue. As a further means of preventing removal of the tube by the patient, the movements of the elbows should be prevented by applying light splints to the entire arm. The tube should be taken out three or four days after introduction, especially if the temperature and respiration be normal.

When the tube is anchored, as mentioned above, extubation is a most simple procedure, infinitely so when compared with that in which the regular instrument or extubator is used. By making taut the string, or wire, with the fingers of one hand, and rapidly passing the index finger of the other hand along the string to the larynx, the tube can be lifted out in a few seconds, and with practically no disturbance to the patient. This method possesses all advantages over the old one with the extubator; principally it does away with the manipulation which the use of the extubator entails, and also the removal of the element of nervousness, which is quite a feature in some cases. In removing an anchored tube it is not necessary even to take the child from its bed, and very often it can be done during sleep, and without the child's knowledge.

In removing the tube it is well to give consideration to the hour and the day; a sunny day is to be preferred, and an early hour in the forenoon, as it is at night that respiratory difficulties are more prone to develop. In some cases, that is those that do not appear favorable after extubation, it is worth while to use steam and stupes, as already described in our efforts to overcome the dyspnea. A return of the dyspnea

may require a reintubation. The tube should then be removed, after three or four days more. In all cases of intubation the utmost gentleness should be observed to prevent damage to the soft parts. After intubation the foot of the bed should be elevated to facilitate drainage, and thereby to lessen the chances of pneumonia.

As said before, intubations should be done only after every effort has been put forth to avoid it. A tube in the larynx acts as a foreign body, causing increased and retained secretions, and in some instances ulceration. Should the child, after repeated efforts, be unable to do without the tube, then a specialist should be called in; what is better would be to send the child to the specialist after release from quarantine. In the handling of tube cases special efforts should be directed towards securing a large, sunny, airy and warm room.

Tracheotomy. The technic for the operation of tracheotomy for the relief of laryngeal stenosis in diphtheria is the same as is that for any other similar condition. Ether anesthesia is used, save in emergency cases, in which an anesthetic will not be necessary. The operator must be sure to make the incision sufficiently long, say 1½ to 2 inches (4 to 5 cm.), and to divide two or three of the rings of the trachea. He should not be in too great a hurry to insert the tube, but should hold open the wound with dilators, or with the handle of the knife held at right angles to the incision, until the breathing is tranquil and easy. In the meantime, he should search for pieces of membrane, and, should any be found, should remove them with forceps. The tube should then be inserted. If there be much mucous or hemorrhage, the foot of the bed or table should be elevated to facilitate drainage from the larynx and trachea. A simple dressing of sterile gauze is placed around the tube and over the wound, and is protected from the secretions of the wound by a flap of rubber tissue.

If the breathing be disturbed, as will be the case in certain instances, because of dried mucus plugging the opening of the tube, the removal of the inner tube and its cleansing will be all that is required. As long as the breathing is without embarrassment the tube should not be disturbed. The changing of the dressing once or twice in twenty-four hours will be all-sufficient.

If a warm room be obtainable, steam will not be required. If this cannot be had, then steam, as from the croup-kettle, and so placed that the air currents will carry the vapor towards the patient, will be sufficient to moisten and warm the room.

A good sign in these cases is a return of the temperature to normal, and a lessening in the respiration rate. This means that the patient has escaped bronchopneumonia, which is the complication most to be dreaded. A bad sign is a profuse discharge of thick, tenacious mucus.

After allowing sufficient time for the separation of the membrane, and for the subsidence of inflammation, an attempt should be made to remove the tube by plugging the opening. If the patient then be able to breathe well, the tube may be removed, and the wound covered with a plain gauze dressing. Sometimes the tube may remain in for days, weeks, or months even. In some instances nervousness will prevent the child from being able to do without the tube. In such cases the idea is to use a closed tube or dummy sufficiently large to fill the wound or opening, but not of sufficient length to reach into the trachea. Should the case promise to be a chronic tube-wearer, then a specialist, as above mentioned, should be consulted.

The complications of diphtheria most important as concerns frequency and gravity are cardiac failure, the paralyses and bronchopneumonia.

The treatment of cardiac failure in diphtheria does not vary, whether this condition be early or late, or whether it be considered primarily myocardial or nervous in origin. The use of alcohol and strychnin as a routine may serve to prevent heart failure. When, however, it occurs, the means at hand for its treatment, while not limited in number, are all of doubtful efficacy.

The patient must be kept absolutely at rest, and even the slightest movement requiring exertion must be avoided. If the patient be not in a state of collapse, the use of such stimulants to the heart as atropin and caffein may steady and strengthen the heart-beat, and avert the danger threatened. If the patient be in collapse, these are also the drugs of choice. Digitalis should never be given. Very small doses of mor-

phin may be of aid, in conjunction with the cardiac stimulants, and help to keep the patient quiet. If vomiting accompanies the collapse, feeding by mouth must be stopped, and rectal feeding instituted. Iced champagne will sometimes be retained, and will help as a stimulant if the stomach tolerates it.

In connection with cardiac failure we must bear in mind that, whereas its prognosis is almost always fatal, yet its prevention is easy. The early administration of sufficient antitoxin will prevent the toxemia that causes both the myocarditis and the nerve degeneration giving rise to heart failure in diphtheria.

This having been done, we have but to keep the patient quiet, to see that he avoids exertion, even the very slightest, and as a routine give small doses of strychnin, $\frac{1}{40}$ grain (0.001 Gm.), every fourth hour, to an adult, and alcohol well diluted, from 1 fluidrachm (3.75 mils), for a child of 4 years, to ½ fluidounce (15 mils) for an adult every fourth hour.

Bronchopneumonia is one of the very frequent causes of death in diphtheria. It occurs principally in cases of the laryngeal type, and more particularly in those that have been the subjects of intubation or tracheotomy. Beyond the securing of a large, warm, sunny and airy room, there is nothing more needed than would be for cases of bronchopneumonia developing from other causes. In some cases I have employed bacterins, and with apparently good results in a few instances. I regret that I cannot speak more positively concerning this.

Post diphtheritic paralysis, due to nerve degeneration caused by the toxemia, generally comes on after the second week of diphtheria. Just as practically every ill effect of diphtheritic toxemia may be avoided by the correct and early use of the antitoxin, so may the palsies be kept but part of the symptom syndrome.

The soft palate is most frequently the site of palsy, which supervenes comparatively early. It makes itself evident by difficulty in swallowing, by regurgitation of fluids through the nose, and by a nasal twang to the voice.

Paralyses of the extremities, eye muscles, sphincters and respiratory muscles also occur. If the last-named condition be severe, death will result.

In severe cases of palatal palsy, nasal feeding is called for until function is restored.

There is, except prevention, no effectual treatment for post diphtheritic paralyses. Unless, as in cardiac failure and the very grave respiratory palsies, death occurs, spontaneous recovery is the rule. No form of medicinal or electric stimulation has ever affected the course of these conditions, in my experience.

The *nephritis* of diphtheria rarely takes a severe course, and beyond the free use of potassium citrate, correct diet, and the careful use of laxatives, little is required. Uremia after diphtheritic renal conditions is very rare.

In mixed infections of diphtheritic sore throat, *cervical adenitis* sometimes occurs. The use of hot fomentations will either abort them or render the glands fit to be opened.

As far as the membrane of the diphtheritic process itself is concerned, I advise the use of no local treatment, with the exception of a mild gargle for adults and larger children.

The treatment of carriers or convalescents ready for discharge, except for persistent positive cultures, is one of the most annoying problems connected with the management of diphtheria. All manner of germicides and antiseptics have been tried, and with indifferent results. The excision of diseased or enlarged tonsils will be found of decided advantage in almost all cases. The removal of adenoids in nasal cases may prove of service. Care should be taken, however, not to undertake operation until all danger of heart or nervous complications shall have passed.

Diphtheria cases may be considered well, and fit to discharge, when all local evidences of the disease shall have disappeared, and when at least two successive negative cultures on successive days shall have been obtained. The physician must be guided by the quarantine regulations of his own locality.

ERYSIPELAS.

This condition is, in reality, an infection by a streptococcus—the *Streptococcus erysipelatis*. Because of the peculiar characteristics of the symptoms, and, again, because of its

frequent localization to the face, it is customary to describe this form and to treat it as a distinct entity.

There is always a portal of entry, frequently a mere abrasion, which is frequently so inconspicuous as to attract no notice. The first symptoms are chill, high fever, the appearance of a red area somewhere over the surface of the body, with, subsequently, a rapid spreading of this area. The affected part is red, swollen, indurated and painful. The edges of the erysipelatous area are sharply marked off by a ridge of indurated skin contrasting markedly with a healthy white skin on the edges.

Soon the surface of the skin is covered with blebs of varying size, filled with clear serum. These blebs later rupture and the surface becomes covered with a crust. Occasionally there is suppuration of the underlying connective tissue. The mucous membranes may be affected; occasionally, meningitis occurs. The writer has seen two such cases.

The fever rises rapidly, often reaching a point between 103° and 105° F. (39.4° and 40.5° C.).

The action of the heart is rapid, the pulse full and bounding. Often the patient is delirious.

Facial erysipelas often begins with the infecting area on the bridge of the nose, both cheeks are rapidly implicated, and very soon the whole face is swollen, the eyes are closed, and the ears stand out as swollen red excrescences on the side of the head. The scalp and neck are often affected. The lesion on the face can scarcely be mistaken for any other condition. On the limbs and about the joints collections of pus may, unfortunately, be mistaken for erysipelas. The differentiation may be made by careful observation that the skin is not affected alone, but that the underlying tissues are likewise involved. Fluctuation is present in suppuration. It is rarely present in cases of erysipelas, and never appears early.

Erysipelas is transmissible from one individual to another, and the early institution of prophylactic measures are essential. All discharges should be collected on material which can be immediately destroyed by burning. The hands should be thoroughly cleansed and disinfected immediately upon handling the case. Because this particular type of streptococci is peculiarly liable to become implanted in wounds and on the

genital mucous membranes, it is highly important for the physician to refuse to attend cases of labor while he is attending a case of erysipelas. If circumstances make it imperative, however, that both sorts of patients be attended at one time, it is necessary that the physician should use gloves while handling the erysipelas case; that before attending a labor case the physician should change his clothing, take a full bath, carefully disinfect his hands before going to the bedside, and use sterile rubber gloves while in attendance on the lying-in woman. These precautions will probably make the labor case safe.

TREATMENT.

Local treatment is of use. The number of applications which have been suggested is almost beyond computing, showing that there is no application which is curative, though several are useful. Ordinary cold water applied by compresses gives comfort, but it does not limit the inflammation.

A saturated solution of magnesium sulphate is of value. It gives the same relief as the water, and, I believe, limits to a certain extent the extension of the inflammation.

Lead-water and laudanum are of value in giving relief to the burning and, perhaps, limit the inflammation.

Ichthyol has been used by the author with no more success than salt solution, magnesium sulphate, or simple water. It is dirty, smells badly, and is scarcely worth the money spent on it.

The edges of the inflammation and the sound skin have been painted with full-strength tincture of iodin, with seeming good results. Phenol has been injected along the advancing edge of the eruption, and good results are claimed for it.

In making use of any of the topical applications, it is well to keep the entire infected surface protected from the air.

There is no specific treatment for erysipelas. Restlessness and delirium may be controlled by full doses of bromid of potassium, 30 grains (1.95 Gms.) every two hours, and then in lesser quantities. This may be reinforced by the use of morphin and hyocin hypodermically.

Tincture of chlorid of iron has been used for years in 30-drop (1.8 mils) doses, well diluted, every three hours. It is

irritating to the stomach, and this complication must be looked out for. Some practitioners give it in connection with quinin. The author has rarely used quinin, and has not seen particularly good effects when he has. Alcohol may be used in small quantities as a food.

Food is important, milk reinforced with cream and sugar, in the form of junket, with cornstarch or other carbohydrates being useful. Large quantities of water should be given to lessen the toxemia.

The heart must be sustained with digitalis, strychnin and caffein, as in other septic conditions. The temperature may be controlled by sponging or by tubbing. As the duration of the illness is relatively short, this antipyretic treatment is not as necessary as in long-continued sepsis.

TUBERCULOSIS.

The several types of tuberculosis, which will be considered here, are general or miliary tuberculosis, tuberculosis of the joints, tuberculosis of the peritoneum, tuberculosis of the genito-urinary organs, tuberculosis of the glands and tuberculosis of the nervous system.

The general treatment of all these conditions must be much the same—rest, an abundance of fresh air, and an abundance of food are necessary to combat the tuberculous tendency, whether the disease is to eventuate in a general tuberculosis, a tuberculosis of the lungs, or a tuberculosis of one of the other organs.

It is a well-known fact that tuberculosis generally is acquired at a very early age, and therefore, one of the first duties of parents and guardians of children is to see that they are not exposed to tuberculous infection any more than possible. If the young of to-day were allowed to have more fresh air in their sleeping rooms, were encouraged to live more in the open, were not taught to be afraid of catching cold—were taught that if they are exposed to extremes of temperature their body should be clothed, so that they would not be chilled and open themselves to the development of any infection, including tuberculosis, there certainly would not be so many

cases of tuberculosis of the lungs, and of the various other organs in later life, according to the statistics shown.

General Miliary Tuberculosis. This generalized tuberculosis occurs in two forms, the typhoid and the meningitic.

In the typhoid form the disease has many of the characteristics of typhoid fever. Certain differences marked are noted between the clinical pictures of the two infections. The patient evidences malaise, becomes the subject of fever, which in the very beginning is usually much more irregular, and may be more intermittent, than in typhoid fever. The pulse, too, has a certain irregularity, sometimes slow, sometimes fast, which is not characteristic of typhoid fever. The intestinal disturbances are not so common in general tuberculosis as they are in typhoid fever, diarrhea being rare, rather than the rule.

The patient also emaciates much more rapidly than is the ordinary rule in typhoid fever.

Examination of the blood is of the greatest value. In typhoid fever there is a positive Widal reaction. In typhoid forms of tuberculosis the Widal reaction is wanting. There is the same leucopenia in both cases. Headache is not so severe in tuberculosis as it is in typhoid fever. Often after a week or two the lungs become the seat of miliary tubercles, and these begin to give the symptoms of general irritation of the chest. It is a sort of granular breathing, and a general bronchitis rather than the sign of any actual consolidation.

Careful examination of the eyes by an expert may show tubercles in the choroid. Spinal puncture may show tubercle bacilli in the spinal fluid; more commonly the cytologic formula shows high lymphocytosis.

The treatment of this universally fatal form of tuberculosis is necessarily without value, so far as saving the patient's life is concerned. Much can be done, however, to make the patient comfortable. The temperature can be controlled by sponging or by bathing; headache may be relieved by the use of opiates, the bromids and chloral, care being taken that the individual is not narcotized. The patient's nutrition may be improved by the use of large doses of milk, and the milk should be prepared so that it is not obnoxious to the patient. It may be flavored with salt or any kind of flavor, or it may

be given in the form of junket. There is, of course, no necessity for the use of a simple liquid diet, but if the patient can, and will eat, foods such as toast, junket, potatoes, cereals and finely chopped beef may be given.

As a prophylactic measure in this form of miliary tuberculosis, indeed, of all other forms of tuberculosis, careful search must be made of the body for tubercular foci. Accessible tuberculous glands should be removed, and if there is a tuberculous joint, this should be treated in the proper way. Tuberculosis of the genito-urinary organs should be cured, if it is at all possible; indeed, every precaution taken to rid the body of any focus of tuberculosis, for a seemingly harmless tuberculous area may very easily light up, and the individual become the subject of a general fatal tuberculosis without warning.

Pulmonary Form of Miliary Tuberculosis. In this form of tuberculosis the symptoms are largely pulmonary from the start—there is cough, and very rapid breathing, with the physical signs of a general bronchitis. Percussion sounds are negative as to signs of consolidation. From the very beginning there is a high degree of cyanosis, entirely out of proportion to the physical signs in the chest. The temperature is irregular, and the pulse is very feeble. Occasionally, but rarely, the sputum contains tubercle bacilli.

The same measures may be employed here for the relief of fever and pain as in the typhoid form. The use of opium for the control of the cough is much more justifiable than it is in cases of any curable pneumonic condition. The case, when the diagnosis is properly made, is necessarily fatal, and all symptomatic relief should be given to the patient, even to the point of mild narcosis by proper drugs.

Fresh air is often of the greatest moment in giving relief to the breathing. Occasionally, oxygen is of great value to relieve, temporarily at least, the cyanosis.

Tuberculous meningitis is a form of miliary tuberculosis which is extremely common, particularly in children. The disease may begin with a convulsion. One convulsion may succeed another, till the death of the child supervenes in a very short time, but this is not the rule. Usually there is a malaise, an irregular fever, the same irregular pulse as occurs

in typhoid forms of miliary tuberculosis, then rapidly beginning meningeal symptoms, persistent headache; a very marked tache, which is extremely common; signs of meningeal irritation as evidenced by paralyses; paralysis, sometimes fleeting, of the ocular muscles; stiffness of the neck, Kernig sign, Babinski sign, and then convulsion, stupor and coma end the scene. When these conditions first appear the spinal canal should be at once tapped, in order to make a diagnosis as to the condition of the meninges. As the case proceeds, the sight is soon lost, the child develops conjunctivitis, the neck becomes markedly stiff, and paralyses of other muscles may occur. The child gradually sinks into coma, Cheyne-Stokes breathing may occur, and usually does supervene long before the end happens.

As far as the writer knows, tuberculous meningitis is always fatal, but much relief can be given toward the cessation of the convulsions and the occurrence of head pains, by systematic tapping of the spinal canal. Here the relief of pressure will often stop the convulsions, and sometimes the headache, which has been extremely severe, will be made to disappear entirely by daily tappings of the spinal canal. In young children, where the fontanels are open, a very marked depression of the fontanel can be seen after tapping, and coincident with this depression convulsive seizures, whether they be general convulsions or not, often disappear.

Tuberculous Adenitis. It has been proved that practically all children over 3 years of age have tuberculosis, usually in the lymphatic glands. Of course, many of these cases of tuberculosis are not at all possible of diagnosis during life, but tuberculosis of the lymph-glands, giving rise to an inflammation of these nodes, is often the very earliest stage of tuberculosis in any form. The lymph-nodes in the neck are perhaps the most commonly affected. Generalized tuberculous adenitis is quite common. Sometimes this occurs as an acute disease, the glands being enlarged over all but the palpable surfaces of the body within a very short time. The disease in this form may run a very short course, so far as fever is concerned, and the glands gradually disappear, or become much smaller.

The enlargement of the mesentery glands, tabes mesenterica, is quite a common affection, and is particularly fre-

quent in young children. The enlargement of these glands gives rise to a very marked abdominal mass, ascites sometimes occur, and the subject rapidly emaciates.

Tuberculosis of the bronchial glands, or the tracheobronchial glands, is quite common, and sometimes gives rise to marked symptoms. Occasionally, pressure on the trachea, or on the bronchus, gives rise to stridor, and harsh cough, which is very constantly mistaken for whooping-cough. The writer has notes of one case where the glands of the posterior mediastinum were large enough to make pressure on the esophagus, and cause an absolute obstruction of the gullet.

Cervical adenitis is the one form of tubercular inflammation of the glands which offers a chance of cure by surgical interference. When these glands are enlarged frequently they become inflamed, caseous in the center, and often give rise to large abscesses in the neck, which, if undisturbed, rupture through the skin, continue to suppurate for a long while, and finally produce unsightly scars. When the cervical lymph-nodes are enlarged and evidently tuberculous, the best treatment is unquestionably removal of these glands before they suppurate, so that the entire chain of glands may be removed without a very great likelihood of their returning.

If great objection is made to the removal of the glands, the suppurating glands may simply be incised and carefully drained, although this is not a good surgical procedure. All the time these glands are enlarged, and during the process of suppuration, the patient should be under the best hygienic conditions, living out-of-doors, given an abundance of food, and a general tonic medication. The best medication to give under these occasions are the syrup of iodid of iron, together with strychnin in proper doses, but I repeat that the glands had best be removed early in the course of enlargement.

The treatment of tabes mesenterica, of necessity, is one of hygiene and feeding. The effect of fresh air and an abundance of food upon tuberculous children, who are virtually at death's door, is scarcely believable. Finally they may be relieved of many of their acute symptoms by simple hygienic and medicinal treatment much like that used in cervical adenitis.

The same remarks may be applied to tracheobronchial glands which give symptoms. Here, on one or two occasions, the use of *x*-ray seems to have caused a retrogression of the size of the glands, which has brought about actual symptomatic cure.

The use of tuberculin in enlarged lymphatic glands is, perhaps, occasionally followed by good results. The administration of tuberculin here, in the writer's experience, is best done by beginning with a very small dose, about $\frac{1}{1000}$ of a milligram, and very gradually increasing the dose, being careful to avoid any reactions. A course of tuberculin may be continued for two or three months, and then an intermission taken, and after four or five months more a second course may be given, often with good results.

This use of tuberculin applies to the treatment of tuberculosis of any part of the body. It should, however, never be used to the exclusion of hygienic methods and good feeding.

Tuberculous Pleurisy. It is claimed by some writers that all attacks of pleurisy are tuberculous in origin. To the writer's mind this is probably an exaggeration, but the number of cases of fibrinous and serofibrinous pleurisy, which are tuberculous, is unquestionably very large.

The symptoms are those of a pleurisy arising under any other conditions—fever, pain in the side, physical signs of a friction sound in the beginning, and afterwards signs of a liquid in the chest. Frequently the onset of this pleurisy is extremely insidious, the patient having little or no pain, and presenting himself to the physician simply with dyspnea. Examination will reveal the collection of a large amount of pleural effusion, or it may be that there has been no effusion, inasmuch as the pleurisy has been largely fibrinous in type, and the movements of the affected side of the chest are very greatly limited by the formation of this new fibrinous material.

The treatment of tuberculous pleurisy, in the acute cases, consists of rest in bed, strapping of the side, and the administration of opiates, if the pain is very severe. If an exudate occurs, as it very frequently does, perhaps the best treatment is tapping, precautions being taken that this slight operation be

done under strict aseptic precautions, and that the site of the aspiration first be sterilized, then made anesthetic by the use of a weak solution of cocain or novocain, ½ per cent. solution being the proper strength, the skin first being infiltrated, and then the underlying tissues. . Through this anesthetized area a slight incision should be made with a scalpel, and through this incision the trocar and cannula can be plunged, practically without pain to the patient. With these necessary precautions a chest may be tapped without the slightest bit of danger to the patient.

In some cases of undoubted tuberculous pleurisy with effusion, the effusion recurs very frequently after each tapping. The question then arises how often such a pleural effusion shall be tapped. The rule which it seems to me proper to follow is to tap three times in succession, each tapping being performed when a mechanical interference of respiration and circulation appear. If the liquid reappears after the third tapping, then tapping should never be done, except when mechanical interference makes it imperative. These patients with a chronic recurring pleural effusion must be put upon all the treatment that is necessary for a general tuberculosis—rest when they have fever, with an abundance of food and abundance of fresh air. As a rule, if this method of tapping is carried out, the effusion will reach a certain size, and give no signs of mechanical interference for many weeks, and sometimes for months. Finally, they cease giving rise to any mechanical effect, because doubtless there is a formation of fibrous tissue to the exclusion of liquid.

Tuberculous Peritonitis. Tuberculous peritonitis may occur as a miliary process in the course of a general miliary tuberculosis, and under these conditions it is usually quickly fatal. However, the tuberculous process may affect particularly the omentum and the parietal peritoneum, and often gives rise to large masses, which can be easily palpated, and frequently these masses are accompanied by an effusion. This effusion is very likely to be not free in the peritoneal cavity, but connected between bands of fibrinous tissue which are formed on account of this process. Sometimes there is very little effusion, and the intestines are bound into one mass of thick fibrous exudate.

The symptoms are abdominal pain, tenderness on palpation, the formation of masses in the abdominal cavity, and often the presence of liquid. It is a remarkable fact that certain cases of tubercular peritonitis seem to recover absolutely by simple opening of the abdominal wall. But it has been observed that frequently these apparent cures due to opening the abdominal wall are only temporary, and this procedure certainly should not take the place of general hygienic treatment.

A patient with beginning tuberculous peritonitis should be put to rest under all the proper hygienic surroundings suggested for tuberculosis of any other organs.

During this treatment careful search must be made for tuberculous foci, such as tuberculosis of the appendix, of the fallopian tubes, and, indeed, for these foci in any portion of the body. If they are accessible, they should be removed, and the general hygienic treatment continued. If, in spite of general treatment, an effusion into the peritoneal cavity occurs and continues, a laparotomy is certainly indicated, and, as already stated, drainage by a simple laparotomy often will cause a cessation of the active progress of the disease. It seems to me, in spite of the statistics which show that tuberculosis of the peritoneum after the abdomen has been opened, tend to relapse, or tend to become chronically ill, the indication for laparotomy is rather to do it early than late. Certainly any procedure which is followed by a cessation of the local inflammation, as laparotomy in tuberculous peritonitis very frequently does, is worthy of an early trial, and should not be used as a last resort.

Genito-urinary Tuberculosis. Tuberculosis of the Bladder gives simply the symptoms of cystitis. It can be positively diagnosed by a cystoscopic examination of the bladder, and the discovery of tuberculous lesions in the bladder wall.

The treatment, first of all, must be a general treatment of the tuberculous condition, and then local conditions best applied by an individual skilled in cystoscopy. As this condition is practically always secondary, and usually secondary to tuberculosis of the kidney, at the time that the diagnosis of tuberculosis of the bladder is made, the ureter should be carefully examined. Usually at the ureteral orifice there is

irritation, and sometimes actual inflammation showing that the disease is primary in the kidney.

Tuberculosis of the Kidney. This disease, not rarely primary, gives rise to symptoms of cystitis, with much pus in the urine. Sometimes there is local tenderness over the kidney, and an enlarged kidney can be discovered, but long before the time of the renal enlargement and a conversion of the kidney into masses of tubercular foci, the diagnosis should be made. This diagnosis should be made by a routine examination of the bladder by one skilled in the use of the cystoscope in every case of cystitis, or with the symptoms of cystitis. If the bladder is found to be free, if a ureteral opening is found to be irritated, and this has the appearance of a tuberculous lesion, then the ureter on the affected side should be carefully catheterized, and the urine from that side be examined for tubercle bacilli. If, after catheterization of the ureter on the opposite side the other kidney is found to be functioning and entirely free of tuberculosis, then certainly surgical removal of the affected kidney is the proper procedure. This should be done, however, only after consultation with a cystoscopist skilled in the examination of the kidneys by the ureteral catheter.

Electrotherapy and *x*-ray have been suggested in the treatment of tuberculous diseases of the genito-urinary organs, just as it has been suggested for the treatment of many diseases in most other parts of the body, but if the disease is limited to one kidney, surely that kidney should be removed, rather than to attempt the removal of the disease by an uncertain process like the use of electricity.

SYPHILIS.

Syphilis is transmitted from one individual to another by means of material containing the specific organism, the *Treponema pallidum.* This micro-organism, as is well known, is a spirochete, and is the sole cause of syphilis, a disease which is either acquired or congenital. When acquired, it is transmitted usually through sexual intercourse, although the disease is not always venereal, and may be transmitted by other means, by kissing, by transference of the virus through drink-

ing cups, towels, penholders, indeed by the medium of any article which has been infected by a syphilitic, and then used by a non-syphilitic.

Congenital syphilis is transmitted from the mother to the child. The mother may be the innocent means of transmission of syphilis, the father having the active disease, but the mother is always infected, as shown by the presence of a Wassermann reaction in her blood, though she may give no symptoms or signs of active syphilis. The congenitally syphilitic child transmits the disease to a nurse when she is allowed to suckle it; indeed, the syphilitic child may transmit the disease to healthy individuals when the active lesions are handled by the nurse, or the exudation from these lesions are in any way permitted to contaminate the mucous membranes or abraded skin of non-immune individuals. For purposes of convenience, and for description, syphilis is divided into the primary stage, the secondary stage, and the tertiary stage.

All three stages of syphilis are transmissible, the primary and the secondary stages much more readily than the tertiary stage, although the last named is certainly transmissible, as shown by inoculation experiments. From practical experience, however, it is proven that in this tertiary stage the disease is rarely transmitted from one human being to another.

The primary stage is represented in the time from the appearance of the chancre to the beginning of the secondary symptoms. This primary stage lasts approximately from six weeks to three months.

The secondary stage begins with the appearance of fever, (which may last during the entire stage if the case is untreated), sometimes closely resembling typhoid fever. Other manifestations at this phase of the disease are eruptions on the skin and mucous membranes, with anemia, general adenitis, sore throat and arthritis.

There is no sharply drawn line between the secondary and tertiary stage, but the tertiary stage is characterized by special lesions of the skin, bones, nervous system and viscera.

There is no tissue of the body which at one stage or the other cannot be affected by the lesions of syphilis.

TREATMENT.

As prophylactic safeguards, public and personal hygiene are of the first importance in dealing with syphilis. The laws which make it legal to establish houses of prostitution, so far as the author knows, have failed to control the disease, syphilis being about as frequent in controlled districts, or countries where there are controlled districts, as it is in other uncontrolled countries. The most helpful means of controlling the disease is probably by a campaign of education. However, this campaign of education must certainly be conducted in a manner which lacks all the elements of hysteria. It would seem to me that education which begins in the school would tend rather to the initiation of venereal habits than otherwise. It is certainly correct, however, that individuals at the time of puberty, or near the time of puberty, should know that there is such a disease as syphilis, and that it is transmitted by sexual intercourse, that it may be transmitted in other ways, and they should be taught the actual physical dangers of illegitimate intercourse, as well as the moral deterioration which comes through such practice. They should know that the danger involves not only themselves, but those with whom they are in intimate contact, and their unborn children.

As I have said, the manner in which this knowledge shall be taught to adolescents is a subject which need not be taken up here, but it is doubtful if it can be safely and properly done through public lectures to the young, which are now so popular. We, as physicians, owe it to our patients, however, when we are asked how they can prevent syphilis, to give them instructions which will protect them, at least, to a certain degree. Of course, the rule which is certain to prevent the spread of the disease, in the large majority of cases, is to abstain from illegitimate intercourse, or intercourse with anyone who is known to be affected, or may be suspected of being infected with syphilis. This, of course, would rule out the great majority of cases of syphilis. However, such advice is rarely heeded, and one is therefore forced to give other rules. The first is that the general health of the individual should be kept up to the highest tone. Second, that inter-

course must not be held with those who are suspected of being syphilitic. Third, if they do not choose to follow this last rule, then they may be told that cleanliness of the genital organs, immediately before and immediately after sexual intercourse, will prevent a certain number of cases of infection. If the genitals are covered with a 30 per cent. calomel ointment this will prevent a certain number of cases. Immediate attention to any suspicious sore on the genitals after intercourse will also prevent a number of cases of infection developing into an actual luetic infection.

Of all these means of prevention, unquestionably the first, to forego illicit intercourse, is the proper one, and the one that we, as physicians, should insist upon. Perhaps physicians are not thought to be necessarily mentors to our patients' morals, but it strikes me that if there is one duty which is incumbent on the physician it is to see that at least by advice and by example his patients shall be taught that a clean and moral life will lead to health. As I said before, this advice is very generally disregarded, but it certainly should be given, and given earnestly, and then his duty in this respect will have been accomplished by the physician.

Since the discovery of the infecting micro-organism, it is known that by certain measures it may be seen in the primary sore. Every primary sore should have the exudate from it carefully examined by an expert in the use of the dark field microscope, so that a very early diagnosis may be made. To wait for the typical characteristic secondary symptoms is to wait until the disease has become generalized, and then treatment is much less efficacious, and necessarily more prolonged. Excision of the primary sore when it is diagnosed as a chancre should be done at once; the earlier the better the chance of limiting the infection. Where practicable, an immediate dose of neosalvarsan or of salvarsan should be given, to be followed immediately by the efficient administration of some form of mercury. A word of caution in regard to the use of salvarsan and neosalvarsan. The intravenous method is unquestionably the most efficient means of administering this most useful drug. However, intravenous medication at the best is not entirely free from danger, and in my opinion intravenous injections should be given only by those skilled in

such procedures, and no one who has not had proper instructions in intravenous injections of salvarsan should attempt it. There is much danger of phlebitis, infiltration of the tissue, and sloughing of the parts under certain conditions. When the administration is properly done, these accidents do not occur. The following is the method used by Prof. A. C. Wood.

The safe administration of salvarsan demands the strictest attention to certain fundamental principles:

1. Scrupulous asepsis must be maintained in every step of the preparation of the solution, and in the administration of the drug.

2. Careful attention to the printed instructions contained in each package.

3. The water used in making the solution, and the salt solution, should be freshly distilled if possible. If not freshly distilled, it must be, at least, thoroughly boiled and free from any particles or sediment.

4. The operator must be certain that the needle is in the vein, before permitting the solution of salvarsan to flow. If any of the drug escapes into the tissues about the vein, a very painful, dense induration results, or there may be extensive sloughing of the tissues. The induration resulting from the salvarsan persists for months.

The method of administration advised by Ehrlich was to use 50 mils (1.7 f℥) of the sterile, freshly boiled water for each 0.1 Gm. (2 gr.) of salvarsan prepared. There is a growing tendency to dissolve the drug in a much smaller amount of water. Some authorities administer the maximum dose, *i.e.*, 0.6 Gm. (10 gr.) in 30 mils (1 f℥) of water; which is introduced by the glass syringe of the Luer type. The latter method simplifies the amount of apparatus and the administration very greatly, but it may still be open to question whether it should be allowed to generally supersede the technic employed by Ehrlich.

For those with limited experience the original method is certainly to be preferred. It is desirable to provide two graduated glass reservoirs, of 300 mils (10 f℥) capacity, each being connected, with some 3 feet of rubber tubing to a 3-way cock, to which is attached the hollow needle. The salvarsan solution is introduced into one vessel, and normal salt solution in the

other. All air bubbles must be carefully eliminated. After the needle is introduced into the vein, the salt solution is allowed to flow; and if several mils of the fluid have passed without causing a visible infiltration under the skin, it may be assumed that the fluid has passed into the vein. The cock may then be turned to permit the salvarsan to flow. If during the administration the patient complains of pain at the site of the puncture, or if there is any sign of infiltration, or other reasons to suspect that the needle may have been displaced, the cock should be at once turned to permit the salt solution to flow. In this way one may determine whether or not the point of the needle is still in the vein. If it is not, it should be withdrawn and a fresh puncture made at some other suitable point.

Neosalvarsan is to be administered in exactly the same way as salvarsan, except that the solution of the former does not require neutralizing with sodium hydroxid. On account of its free solubility and its milder irritating qualities, it is more suitable for administration in concentrate solution. The maximum adult dose of 0.9 Gm. (14 gr.) has been administered in as little as 10 mils (2.7 f3) of water. The writer is not inclined, however, to recommend this method for general administration at the present time. Ehrlich recommended the use of 25 mils (6½ f3) of water, or salt solution, for each 0.15 Gm. (2⅓ gr.) of neosalvarsan.

Mercury may be given by the mouth, by inunctions, and by intramuscular injections. By the mouth perhaps the best preparation is the protiodid of mercury. This may be given in ¼-grain (0.016 Gm.) doses three times a day, gradually increasing the number of doses until soreness of the gums is noticed. During the administration of mercury in this or any other form, the mouth is to be kept scrupulously clean by brushing several times during the day, and by using a simple alkaline mouth-wash, either Dobell's solution, boric acid solution, or one of the pleasant proprietary solutions. The liquor antisepticus, of the United States Pharmacopeia, is quite efficient and pleasant as a mouth-wash, and should be used in preference to proprietary preparations of the same character.

After two weeks the initial dose of neosalvarsan or salvarsan should be repeated, again to be followed by a course of

mercury, and this should be kept up until four or five injections of neosalvarsan or salvarsan have been given, the intervals between the doses being used for the purpose of administering mercury. Besides the administration of mercury in the form of protiodid, mercurial inunctions may be used; a drachm (2 Gm.) of the mercurial ointment should be rubbed into the portions of the body covered by thin skin, *i.e.*, the axillæ and the inner aspects of the thighs. These inunctions should be given every day, for six or seven days, and then omitted for four or five days. The rubbing is best done by an attendant, who should have the hands protected by rubber gloves; it should take at least a half an hour to rub in a drachm of the ointment, until it has entirely disappeared from the surface.

Another method of administering the ointment is to put it upon the soles of the feet, and then encase the feet in a thick woolen stocking, but this method is uncleanly, and is not to be recommended as a usual procedure.

The intramuscular injections of mercury is an extremely convenient and efficient way of administering this drug. It may be given in the form of salicylate of mercury, 10 grains (0.6 Gm.) to an ounce of a mineral oil, preferably albolin. This is given in doses of 10 mils (160 *m.*), deep into the muscles. If it is given into the subcutaneous tissue it is not absorbed so well, and it is quite painful, so that a long needle should be used, which will convey the material into the muscle itself. In the place of salicylate of mercury, gray oil, a suspension of mercury in oil, may be used. An extremely carefully prepared mercurial ointment is first made, and then a certain portion of this is rubbed up with pure olive oil. The great objection to gray oil is the difficulty of its proper preparation, and the inadvisability of using a preparation which is not accurately made. These intramuscular injections are given at intervals of two or three days, care being taken that not enough mercury is given to cause severe salivation, and the same rule should be followed as is observed in using mercury internally. When the gums begin to get sore, the administration should be stopped. In severe cases, particularly in cerebral cases, I have used the injection every day, with very good effect. Of course, the time in which the salivation occurred was much shorter,

and had, therefore, to be watched more carefully. There is some danger in this intramuscular injections of mercurial preparation, but it is very slight.

First, abscesses, which can be avoided by strict asepsis.

Second, so-called fat embolism. If the emulsion chances to be injected directly into a vein, the oil will be carried directly to the lung, causing an embolism there. This accident is characterized by pain in the side, bloody expectoration and cough. By using not too large a dose, however, the danger is extremely slight.

After the use of this method of treatment for four to five months, all treatment should be stopped if there are no symptoms, and the patient allowed to go with a simple tonic of iron and nux vomica for two months, when a Wassermann reaction is taken. If the reaction is positive, then the treatment should be renewed. If it is negative, an interval of three months may be allowed to elapse, when a second course of five or six treatments such as described is again given. These intermittent treatments must be kept up for at least three years before the patient can be considered cured, and no person should be pronounced cured until he has had at least two negative Wassermann reactions after the last course of treatment.

Iodid of potassium should be used only in the latter part of the treatment, in the tertiary stage, 10 grains (0.6 Gm.) of iodid of potassium given three times a day is efficient.

Treatment of Lesions of the Tertiary Stage. Every patient under suspicion of syphilis should have a Wassermann reaction made. If this is positive, then active treatment should at once be begun. If the Wassermann reaction is negative, and the symptoms and signs of syphilis are positive, then the active treatment should be begun, in spite of the fact that the blood is negative. Indeed, after the administration of a dose of neosalvarsan or salvarsan, the Wassermann reaction, which was at first negative, may become positive. Where the question is of great importance, and the blood Wassermann is negative, the spinal canal should be tapped and a Wassermann test made of the spinal fluid. This will frequently be positive when a blood reaction is negative.

The tertiary stage of syphilis and those cases where no syphilitic symptoms are present, but a Wassermann is posi-

tive, should be at first treated by intensive use of neosalvarsan or salvarsan, and this to be followed by mercury, very much after the same plan as is used in the cases of secondary and primary stages. Iodid of potassium, where there are visceral lesions, is of the highest importance; 10 to 20 grains (0.6 to 0.2 Gm.) three times a day will cause the disappearance of old ulcers and visceral lesions very promptly.

The action of iodid of potassium is perhaps not very well understood; certainly, it is not the same sort of action as that of mercury; it is not a spirochetocide, and, therefore, it does not act in the same manner, but it is most useful, and should be used in all tertiary lesions.

For the treatment of cerebrospinal syphilis, H. F. Swift and A. W. M. Ellis evolved a method of treatment of this disease by autosalvarsanized serum into the spinal canal. This has been proved by many observers to prevent advance of the disease, and to convert individuals who apparently were fast becoming invalided, into fairly normal condition.

The subjoined method of giving these injections into the spinal canal are taken from an article by Swift and Ellis.

Technic of Subarachnoid Injections.—One hour after the intravenous injection of salvarsan 40 mils (1⅓ f3) of blood are withdrawn directly into the bottle-shaped centrifuge tubes, and allowed to coagulate, after which it is centrifugalized. The following day 12 mils (0.194 *m.*) of serum is pipetted off and diluted with 18 mils (0.292 *m.*) of normal saline. This 40 per cent. serum is then heated at 56° C. (132.8° F.) for one-half hour. After lumbar puncture the cerebrospinal fluid is withdrawn until the pressure is reduced to 30 mm. cerebrospinal fluid pressure. The barrel of a 20-mils (5½ f3) Luer syringe (which has a capacity of about 30 mils [8 f3]) is connected to the needle by means of a rubber tube about 40 cm. long. The tubing is then allowed to fill with cerebrospinal fluid so that no air will be injected. The serum is then poured into the syringe and allowed to flow slowly into the subarachnoid space by means of gravity.

At times it is necessary to insert the plunger of the syringe to inject the last 5 mils (0.82 *m.*) of fluid. It is important that the larger part of the serum should be injected by gravity and if the rubber tubing is not more than 40 cm. long the pressure

cannot be higher than 400 mm. Usually the serum flows in easily under even a lower pressure. By the gravity method the danger of suddenly increasing the intraspinous pressure to the danger point, such as might occur with rapid injection with a syringe, is avoided. Frequently there is a certain amount of pain in the legs, commencing a few hours after the injection. The pain is more often noticed in tabetics than in patients with cerebrospinal syphilis. It can usually be controlled by means of phenacetin and codein. Occasionally morphin is required.

Congenital syphilis is best treated by salvarsan in proper doses when the child is a year old or over. Before that time inunctions of mercury offer the best chance of relief. A syphilitic child should be nursed only by its mother. There is great danger of transmitting the disease to a wet nurse.

The patient will constantly ask the physician when he can get married after he has contracted syphilis. Here, again, the advice of the physician is frequently not followed, but nevertheless, it should be given in good faith to all those who ask it. A patient should not be allowed to marry for at least three years after having contracted syphilis, and after having been under carefully conducted treatment during those three years, and not then unless two carefully conducted Wassermann's are negative. If the person is married, he should certainly be advised to abstain from sexual intercourse with his wife until at least two years have passed. Of course, if the wife is already infected when the patient is seen by the physician, this advice need not be given. Most of the inefficient treatment of syphilis is due to the fact that patients constantly cease treatment after the symptoms have disappeared. Just how to overcome this fault remains for the physician himself in every particular case to decide; but in those who are neglectful, and in those who are intractable, the dangers of syphilis should be fully explained to the patient, and then, of course, whether or not he shall carry out the treatment remains with himself. On the other hand, nervous individuals, particularly if they be married, and if they be intelligent, should be assured that syphilis is a curable disease, and if the treatment is carried out properly, and hygiene persisted in, he will prevent any serious after-effects, and can prevent transmitting it either to his wife or to his children.

GONOCOCCUS INFECTIONS.

There is, perhaps, no disease so little understood by the laity as is gonorrhea. This misunderstanding unfortunately sometimes extends to the ranks of the medical profession. Beginning as it usually does as infection of the urethra in the male, and of the urethra and the vagina in the female, it is considered a purely local infection. The infection is certainly local in the beginning of the case, but very frequently—more frequently than is usually suspected—general infections, a true septicemia, and local infections such as arthritis, diseases of the eyes, ophthalmia and iritis, diseases of the pelvic organs in women, occur as evidence of the gonorrheal infection.

When these infections are accompanied by the local evidence of gonorrhea, which is easily diagnosed, the real cause of the arthritis and other infections may be suspected. When, however, an individual with arthritis, presents himself to a physician for diagnosis, unfortunately, gonorrhea is about the last thing that is suspected by the physician as the cause of the arthritis; and even though the diagnosis be suspected, and confirmed, the treatment of these gonorrheal secondary infections is often far from being satisfactory.

The methods of diagnosis must consist, first, in a careful scrutiny of the history of the case. This is notoriously a very unsuccessful method by which the true cause of the secondary disease can be established, for the reason that those who have had gonorrhea, either forget the circumstance, or are not willing to admit it; and, again, many are not aware that they have had a gonorrheal infection, while they may be at the time suffering from the ravages of that disease.

The methods of diagnosis of gonococcus infection depends, first, upon the recovery of the gonococcus. This may occasionally be done from the prostate in men, from the vagina and urethra in women; and, in certain cases of arthritis, the microorganism may be obtained from the fluid in the joints. In certain cases of general infection it may be obtained from the blood; but these findings rarely are made except when the patient is under treatment in an institution where first-class laboratory facilities are at hand, and used.

Gonorrheal fixation tests have lately been used with some success, although this method is not as yet considered as positive a test for gonorrhea, as is the syphilitic fixation test for syphilis.

The conditions which will be considered under this chapter exclude the local manifestations of gonorrhea, that is, gonorrheal urethritis. For the symptomatology and treatment of this disease, the reader is referred to a work dealing with that particular condition.

It is a well-known fact that a very large percentage of the blind in the world become blind from an ophthalmia at birth, from a gonorrheal infection usually present in the mother. It is also a well-known fact that salpingitis in women, ovaritis, and general pelvic inflammation are very frequently the result of a gonorrheal infection spreading from the local seat of the disease, the urethra and the cervix. Unfortunately this pelvic condition, so frequent in women, is often entirely innocently obtained. A man with an uncured gonorrhea becomes married, infects his wife, who, as a rule, is never aware of the reason of her illness.

The treatment of both ophthalmia and pelvic infections in women, will, like the local infection of gonorrhea, not be treated here, the one condition being best dealt with by the ophthalmologist, the other by the surgeon. However, the practitioner should always bear in mind that when a child develops a conjunctivitis immediately after birth, it should be considered a gonorrheal ophthalmia until it is proven otherwise. If this view is taken by general practitioners who attend women in labor, fewer cases of injury to the eye would occur.

Vaginitis in young children is a very common disease. This condition is very refractory, and the only possible way to treat it is by isolation of the individual patient, and closing the ward during an outbreak of the disease until all the children are perfectly well, and the room can be cleaned and disinfected.

Thayer, Blumer, and Cole have exhaustively studied gonorrheal septicemia, and have described in detail a type associated with endocarditis, febrile cases resembling closely typhoid fever, and cases of general septicemia with multiple abscesses over the body.

Osler reports one case, with small abscesses of the prostate, where the subject died within four days after the initial chill. Cases of puerperal septicemia consequent to gonorrheal infection are well known. The endocarditis, the result of gonorrhea, differs not at all in its physical signs from that which occurs from any other infection, and cannot be diagnosed except by discovering the focus of infection; indeed, as much may be said of the fever resembling typhoid fever, and the deaths from septicemia, and from puerperal septicemia. Unless the gonococcus is discovered in a focal infection, the true cause of the disease may never be known.

Gonococcus Arthritis. Gonorrheal arthritis may occur during the acute attack of urethritis, or during a chronic urethritis, gonorrheal in origin; also it may occur when there is no urethritis discoverable, but with the micro-organism active in the prostate gland.

The inflammation in arthritis is either periarticular or within the joints, which rarely suppurates, but become disabled and distorted. If an individual with a gonorrheal urethritis and prostatitis develops an arthritis, it is very evident that the condition is gonorrheal in origin. This usually may be positively established by withdrawing liquid from the joints, and examining it for the specific bacteria of the disease.

The disease may be polyarthritic or monarthritic, the latter form being perhaps the more common. The affected joints become swollen, extremely tender, and this persists in spite of the ordinary treatment for arthritis usually given. An actual septicemia may occur from the arthritis, particularly when it is polyarthritic.

Unusual joints are frequently affected, such as the sterno-clavicular, the temporal maxillary, and the sacro-iliac. Persistent sore heels, the result of a periostitis, is a very common condition as an after effect of gonorrheal infection. When an individual presents himself with sore heels, with practically no other symptoms, an *x*-ray should be taken to see if the periosteum is affected, and the proper treatment afterward instituted.

TREATMENT.

The treatment of this disease is very unsatisfactory. Before any treatment is instituted, which promises success, the

existence of a gonorrheal infection must be established. This may sometimes be done by careful searching for local infection, and cultivating the gonococcus from it. Sometimes it may be established by culture from the blood.

The treatment of the septicemia must be on general lines. The patient must be kept in bed, and drink an abundance of water. If there has been a local focus discovered, that point of infection must certainly be cleared up before there is much hope of the septicemia disappearing.

Endocarditis must be treated as endocarditis under any other conditions. The patient must be kept in bed with an ice-bag over the precordia, until active signs of fever and leucocytosis have disappeared.

In arthritic form the joints must be fixed, and in a position in which they will be useful after the disease has spent its course, for, as noted above, many of the cases of arthritis leave behind them a deformed joint.

Gonorrheal vaccines and gonorrheal serums may be tried in these conditions. In a case of gonococcal arthritis, a stock gonorrheal vaccine may be used, with some hope of success. Perhaps this is one of the conditions in which a vaccine gives best results.

In the general conditions, a gonorrheal serum may be used, with the hope that the antitoxic properties of the serum will shorten the course of the disease.

In the chronic condition of inflammation of the joints, very gentle massage, with hydrotherapy should be used, with the hope of rendering the joints painless and supple.

INFLUENZA.

Influenza, *la grippe,* is due to infection by Pfeiffer's bacillus, a small, non-motile micro-organism which usually lodges in the respiratory passages first, and at this site elaborates absorbable toxins accountable for the fever, the toxemia, and the systemic symptoms of the disease. Influenza is now endemic in this country, and frequently sporadic cases are reported throughout the country. Since 1890, when the last great epidemic occurred, there have been isolated cases of true influenza, but no marked epidemic. The vast majority of

common colds are not due to influenza, and the exact relation between epidemic influenza and the epidemic colds is a moot point. It is, however, evident, as proven by cultural methods, that most of the epidemic colds are not due to the influenza bacillus. The disease frequently becomes pandemic, and spreads over the country in great waves of disease. In 1830-33, 1836-37, 1847-48, and in 1888 and 1890, a scourge of the disease swept over North America within a few weeks.

The ordinary attack of influenza begins with sudden, rather severe aching of the whole body, severe headache, coryza, laryngitis and bronchitis. The exhaustion is extreme, and out of all relation to the apparent seriousness of the illness; it occurs suddenly, and evidently the toxin elaborated by the infection is formed in great quantities and disseminated with extreme rapidity. With the onset of these symptoms, the temperature rises and reaches a height of 102° F. (38.8° C.) or 103° F. (39.4° C.), and in severe cases reaching as high as 105° F. (40.5° C.). This temperature remains between 102° and 103° F. (38.8° and 39.4° C.) for five or six days, when, in the absence of complications, it falls gradually to normal. During the height of the attack there is severe and unproductive cough, the coryza increases, the sore throat becomes worse, and the aching of the limbs and the headache become almost unbearable. As the temperature falls, the severity of the symptoms diminish, and at the end of about ten days the patient feels well, but is extremely exhausted. This extreme exhaustion is one of the peculiarities of this condition.

Various types of influenza are recognized, depending largely, or altogether, upon the effect of the toxin upon certain sets of organs.

The respiratory, gastro-intestinal, and nervous types are those most commonly observed, and to these may be added the typhoid, circulatory, renal and arthritic types.

The fact that more or less frequently true influenza localizes itself in these organs almost exclusively, gives rise to many errors in diagnosis, especially during an epidemic of the infection. All of us, and particularly the overworked general practitioner, in the hurry and stress of his duties, is very likely to lose sight of the fact that while attacks of influenza

make up the greater number of cases he sees, other diseases can, and do occur, during such times. The consequence is that enough attention is not given to attempts to differentiate influenza from other diseases, and typhoid fever, pneumonia, meningitis, and so on, are frequently overlooked in their incipient stages, and are treated as influenza, much to the detriment of the patient.

Respiratory types of influenza are by all odds the most common, and in such instances the symptoms of bronchitis, laryngitis, tonsillitis and coryza dominate the clinical picture. In this type bronchopneumonia quite frequently supervenes as the direct result of invasion by the influenza bacillus. This influenzal manifestation or complication is very frequently overlooked, but the association of continued fever, leucocytosis, unusual dyspnea, and characteristic physical signs relating to the lungs make the diagnosis certain.

Nervous symptoms during an attack of influenza sometimes dominate the picture. Here extreme headache, actual neuritis, and signs of meningitis are the symptoms most in evidence.

Gastro-intestinal symptoms such as vomiting, diarrhea, and more or less localized abdominal pain are certainly very frequent during an epidemic of influenza. Whether they are simply complications of influenza or due to bacteria other than influenza bacilli, is difficult to prove.

Typhoid forms of the grip also exist, and such types are due to an unusually severe toxemia. Here the greatest danger is of considering every typhoid-like case influenza, instead of making careful blood examinations for the Widal reaction, and searching for typhoid spots, to exclude true typhoid fever.

It is an almost impossible task to isolate cases of influenza during an epidemic, but children and aged persons should certainly be kept out of contact with known cases of the disease whenever possible. As a general measure of prophylaxis the sputum should be burned whenever practicable.

TREATMENT.

There is no specific drug for influenza, and there are no specific measures to be observed in the management of the

infection. Every case, even the lightest, however, should be put at rest in a well-ventilated room, and the patient protected by proper bed-clothing. In every instance the patient should maintain this rest until all of the acute symptoms have passed. Drugs such as the salicylates, especially phenol salicylate (salol) and acetyl salicylic acid (aspirin) should be used for the relief of the muscular pains and the headache. The bowels should be kept active with calomel, followed by some mild saline cathartic, such as citrate of potassium or sodium phosphate. Active purging is weakening and not desirable. A mixture of citrate of potassium and sweet spirits of nitre is useful as a sudorific.

Severe headache is frequently relieved by caffein (the alkaloid) and sodium bromid. Two grains (0.13 Gm.) of the former and 15 to 20 grains (0.972 to 1.3 Gms.) of the latter every three hours is an appropriate dose for an adult. In the face of a persistent neuralgic headache, and when there is tenderness over the sinuses, a sinusitis should be suspected and looked for. Indeed, most of the attacks of headache which persist are due to swelling of the lining membrane of the sinuses. By no means always, and, indeed, very seldom, is the sinusitis suppurating. Occasionally there remains for many days after the ordinary headache has disappeared a periodic pain which comes on early in the morning and disappears by mid-day or earlier. This periodic headache is relieved and apparently cured by large doses of quinin. Ten grains (0.65 Gm.) of quinin given on retiring will usually cause this annoying symptom entirely to disappear within the course of a few days.

The bronchitis is relieved by the general measures suggested; it may be further relieved by the use of potassium citrate and chlorid of ammonium. If the cough is frequent and unproductive, paregoric, codein or heroin will give much relief, and often converts a very distressing condition into one which is mild. Care, of course, must be taken to avoid narcotizing the patient.

Otitis media must always be borne in mind as a possible complication, and the ears should always be examined, especially in children. Any bulging of the drum calls for imme diate puncture of the drum-membrane. Mild attacks of mid-

dle-ear disease can be aborted by the instillation of a 5 per cent. phenol solution in glycerin; or atropin, $\frac{1}{150}$ grain (0.0004 Gm.), in the form of a hypodermic tablet, may be dropped into the canal and dissolved *in situ* with a few drops of warm water. The ear should be covered with a pad of cotton.

Laryngitis may be relieved by bromid of potassium and citrate of potassium internally, and by inhalation of steam, plain or medicated with compound tincture of benzoin.

Pneumonia must be treated as pneumonia of any other type. (See p. 28.)

Vomiting can be controlled by the withdrawal of food, and, if it persists, with 10 grains (0.65 Gm.) of bismuth subnitrate in lime-water. The following prescription will be found effective:

℞ Bismuthi subnitratis ..... 16 (246.8 gr.).
Pulv. acaciæ ..... 20 (308.6 gr.).
Liquor calcis .....q. s. ad 120 (4 f℥).
M. S.: One teaspoonful (4 mils) every two or three hours.

A mustard plaster to the epigastrium is often comforting. If the bismuth is not effective, a powder such as the following may be used:

℞ Codeinæ ..... 0.16 (2½ gr.).
Cerii oxalatis ..... 2.00 (40 gr.).
Cocainæ hydrochloridi ..... 0.16 (2½ gr.).
Ft. pulv. no. j.
S.: One powder every three hours.

Care must be taken not to give enough of this powder to produce a toxic effect.

Diarrhea. This demands also withdrawal of food and, perhaps, fractional doses of calomel, every half-hour, followed by a saline, until the offending material is cleared from the bowel. This may be followed, if necessary, by a formula such as the following:

℞ Bismuthi subnitratis ..... 16 (246.8 gr.).
Phenolis salicylatis ..... 8 (123.4 gr.).
Misturæ cretæ compositi ..... 120 (4 f℥).
M. S.: One teaspoonful (4 mils) every two or three hours.

Neuritis. The pain of this disturbing condition is best relieved by fixation of the limb, if one of the distal nerves be affected; by the application of heat in the form of a hot-water bottle or an electric pad; and sometimes by the administration of acid acetyl-salicylic in 5-grain (0.325 Gm.) doses, acetanilid in 5-grain (0.325 Gm.) doses, and acetphenetidin (phenacetin) in the same dosage, the use of which may give relief when the salicylates fail. These drugs must not be continued more than twenty-four hours consecutively, for fear of disintegrating the blood, which may follow prolonged use.

Meningitis. This may show all the symptoms of meningitis due to other micro-organisms. The diagnosis depends upon the discovery of influenza bacilli in the spinal fluid. The patient should be at rest, should have the spinal canal tapped at least every twenty-four hours. Torrey has reported a case of this fatal form of influenza which ended in recovery after the spinal canal had been tapped fourteen times.

Waldstein has perfected a serum for the treatment of influenza which is said to have some curative value. Torrey did not find any particular relief after its use in his case.

To combat active nervous unrest, bromid of potassium in full doses is of value. To this may be added chloral hydrate.

Cardiac Symptoms. Disturbances of the cardiovascular system should be treated by absolute rest. If an actual endocarditis develops, this rest should be prolonged into weeks, in order that the local inflammation of the valves may be as near at an end as possible before the stress put upon the heart is increased by allowing the patient to move about. Ice-bags to the precordia in the early stages and the avoidance of digitalis are necessary.

Urinary Inflammation is correctly treated by the administration of large quantities of water. Hexamethylenamin is useful, if the pelvis of the kidney or the bladder are infected, but stimulating diuretics should not be advised. If there is a complicating nephritis, such drugs but increase the renal inflammation. Strychnin sulphate, $\frac{1}{30}$ grain (0.002 Gm.) three times a day, or tincture of nux vomica, 20 drops (1.25 mils) three times a day, is of great value.

The *typhoid forms* of *la grippe* must be treated by rest, an abundance of good food and strychnin. If the fever is high, cold sponging should be used.

Convalescence is often prolonged. The patient is weak, as evidenced by exhaustion on the least exertion, by breathlessness, and by cardiac palpitation. If the attack has been one of severity, the patient should be kept away from his work for a prolonged period, and when he does return should not work the full number of hours at a time.

CEREBROSPINAL FEVER.

This condition, also known as epidemic meningitis and as spotted fever, is a true meningitis due to infection by the *Diplococcus intracellularis meningitidis.*

The disease is transmissible from man to man, from man to lower animals, and from animal to animal through several generations.

Flexner and his co-workers have shown that the disease can be transmitted from man to monkey by means of the nasal discharge, and they believe that the portal of entry through which human beings become infected is the nasal mucous membrane.

The attack usually begins suddenly, with high fever, intense headache, and, as early symptoms, cervical rigidity, stiff back, Kernig's sign, the Babinski reflex, and Brudzenski's sign are of prime importance. Herpes frequently occurs, and there is always a polymorphonuclear leucocytosis. The patient rapidly grows worse, and in about 75 per cent. of the cases succumbs to the disease, if proper treatment is not instituted at once. Many variations of this ordinary picture occur. The most important diagnostic sign is the condition of the spinal fluid, the presence of the meningococci in it is an unmistakable pathognomonic sign.

The fluid can be obtained only by puncture of the spinal canal, which simple procedure should be done early, and in every case of suspected meningitis. Only by the character of the spinal fluid can the type of the meningeal inflammation be ascertained. Indeed, I think that spinal puncture may be regarded not only as the sole means of making a positive

diagnosis, but here, as in meningitis of any type, as one of the most important therapeutic measures.

The operation is simple, and every physician should be able to perform it. The skin of the back should be cleansed along the spinal column. Iodin should then be applied over the line of the spinal processes and about 2 inches (5.08 cm.) on each side, reaching from the last thoracic vertebra to the sacrum. If the patient is a child or a nervous adult, novocain or cocain should be injected into the skin and deep into the tissues. I believe this lessens the pain of entering the spinal puncture needle. A spinal puncture needle should be of small caliber, provided with a short, beveled tip, and fitted with an accurately ground obturator. The needle is sterilized, the hands of the operator are made as sterile as possible, and, by choice, sterile gloves are used. The patient should have the spine curved anteriorly as much as possible. The second, third and fourth, lumbar spinal processes should be identified, the last named lying approximately on the level of the highest point of the crests of the ilia. The needle then held firmly is plunged in the median line straight forward between the second and third, or the third and fourth lumbar spinous process. It will enter from 2 to 3 inches (5.08 to 7.62 cm.), depending upon the thickness of the tissues of the back. As the needle enters the canal the membranes will be felt to give way suddenly. The obturator is then withdrawn and the fluid flows freely from the needle end. If no fluid comes, most likely the needle is not in the canal, or it is an interspace too low. The dry tap is usually due to faulty technic, although there are instances where the fluid is purulent and too thick to flow, and others in which there is little fluid. Every dry tap made by the author has been due to faulty technic, as proved by the fact that a successful tap has been made shortly after, either by himself or by another operator.

Sometimes it is well to go into the canal from a lateral position. In this event the same site should be selected, but the needle is to be started about 1 inch (2.54 cm.) to one side or other of the midline, and then pushed slightly upward and inward. This gives more pain than when the needle is inserted in midline.

In the vast majority of cases the fluid of cerebrospinal meningitis is cloudy. It should be examined at once by the microscope. If the cloudiness is due to polymorphonuclear leucocytes, the specific treatment should at once be given. If it is practicable to stain a smear of the fluid, the presence of the diplococci within the cells will be seen, and the diagnosis will be certain. But in any cloudy meningeal fluid the specific serum should be given after the first puncture, even if there is no opportunity of making a bacteriologic diagnosis at the time. A bacteriologic diagnosis, however, should be made before it is time to give a second dose of serum.

TREATMENT.

The specific treatment is Flexner's antimeningococcic serum. This should be on hand at the time of tapping every suspicious case, and should be given through the same needle with which the canal is drained. It is wise to use the aspiration needle which comes with the serum. From 30 to 40 mils (1 to 1.3 f℥) of spinal fluid are withdrawn; then, by the gravity method a little less serum is allowed to flow into the canal than has been removed. The aspiration and introduction of serum should be repeated every twelve to twenty-four hours. The fluid, after the second tapping, is likely to be much clearer, and to contain fewer bacteria than that first withdrawn, if the case is a favorable one.

The reason that the serum treatment is fully described is that, like diphtheria antitoxin, the earlier it is given the better the results of the case. To wait a number of hours until a microscopic examination could be made, might mean the difference between life and death.

It is certain that the disease in question is communicable from one individual to another, usually through the secretions of the mouth and nose, and, in view of this, careful prophylactic measures are to be observed. This makes it wise always to disinfect such discharges. The cases should be isolated. In epidemics the cases seem to be more virulently transmissible than in sporadic cases, but the latter are about as readily communicated from the sick to the well as are cases of croupous pneumonia.

Food is important—milk, eggs and cereals should be taken in as large quantities as the patient can stand.

Hexamethylenamin should be given in doses of 5 grains (0.324 Gm.) every three hours, a careful watch being kept meanwhile on the urinary secretion, for this drug irritates the urinary tract, and is to be discontinued when symptoms of irritation supervene. Morphin or opium in some form should be given for the pain and restlessness, or the patient may be placed in a warm bath. Bromid of potassium or sodium is of value, if given in 20-grain (1.3 Gms.) doses every two hours. This may be combined with chloral hydrate in 10-grain (0.65 Gm.) doses.

The fever can be controlled by sponging or by tubbing.

Tapping the spinal canal alone is of use as a tolerably specific therapeutic measure, and the operation should be repeated daily so long as the symptoms either remain stationary or become aggravated.

The postfebrile emaciation should be combated by massage, rest, fresh air and good food.

Arthritic symptoms are best treated by massage, and by the injection of the serum into the affected joint.

TETANUS.

The cause of tetanus is an initial wound in which the bacillus of tetanus becomes implanted, and without such a wound the bacillus does not find lodgment in the human body. Any wound, varying from a mere abrasion to a severe laceration, may become infected with the tetanus bacillus. The bacillus has its habitat particularly in the dirt of the street, in and about stable ground, or around pastures where horses have fed. The micro-organism is anerobic, and, therefore, wounds which quickly heal over after having been infected with the tetanus bacillus are those most likely to give rise to the disease.

Unfortunately, cases of tetanus have followed vaccination against smallpox, but in no instance has a contamination of the vaccine itself been conclusively proved; and in more frequent instances, three of which came under the writer's notice during the last year at the Episcopal Hospital, the tetanus

bacillus had become implanted on the ulcer resulting from the vaccination, entirely independent of the vaccine virus itself.

The disease begins first with stiffness of the jaws, and sometimes stiffness of the neck. This rapidly develops into a tetanic spasm of the muscles, and this spasm quickly affects all of the muscles of the body; first the muscles of the back, and afterward the muscles of the abdomen and limbs. The muscles of the face are particularly affected. Inability to open the mouth, or "lock-jaw," occurs, and during an active spasm the face muscles are drawn up into a grin-like position, the so-called "sardonic grin."

Tetanus neonatorum is simply tetanus occurring in the newborn infant, in which the bacillus gets its lodgment in an improperly dressed umbilical cord.

TREATMENT.

Perhaps the most important point in the treatment of tetanus is the treatment of the initial wound, and the prophylactic use of antitetanic serum, which is of particular moment in wounds received from fire-crackers and blank cartridges, which are so commonly used on the Fourth of July. The wound should be thoroughly opened and cauterized, either with an actual cautery or with nitric acid, and then it should be kept open, in order that any bacillus with lodgment in the wound may be exposed to the air, which is fatal to its life. The individual should then at once receive from 5000 to 10,000 units of antitetanic serum into the muscles. In this way tetanus can practically be overcome.

Until very recently the mortality of developed tetanus was extremely high, and, while still high, there is an appreciable diminution in its fatality. As soon as a patient with developed tetanus is seen he should be given 3000 units of antitetanic serum into the spinal canal, 5000 units into the muscles, preferably the muscles of the buttock or back, and 10,000 units into the vein. This may be repeated from twelve to twenty-four hours after, according to the necessity of the case.

To quote Keen's masterly treatise on "The Treatment of War Wounds," one should "expect tetanus in all wounds and

prevent its onset," and the observation of this rule, originally insisted upon by Gibson, has practically caused the disappearance of lockjaw in the armies now engaged in conflict on the Continent. "This conquest of tetanus is one of the notable victories of the war."

In an article by Matthias Nicholl, read before the Association of American Physicians, in 1915, the intraspinal administration is considered of particular importance, and he reports 20 cases which are treated as they occur in or about the city of New York, with three deaths. This is an unusually low mortality for developed tetanus. His method is to give 3000 to 5000 units into the spinal canal, 10,000 units into the vein, and repetition of the intraspinal dose in twenty-four hours, and a subcutaneous dose of 10,000 units thirty-six hours later.

The ordinary methods of treatment, of course, must be carried out in these cases of tetanus. The patient is kept in a darkened room, free from drafts, and from visitors. He must not be disturbed. Even the physician must disturb him as little as practicable. The reason for this is that frequently a patient may be lying in a relatively comfortable position when he has tetanus, and the least disturbance will throw him into a violent tetanic spasm.

He should be given large doses of bromid of potassium and chloral, the chloral in 10-grain (0.6 Gm.) doses every three hours, and bromid in 20-grain (1.3 Gm.) doses every three hours, which can be kept up for three or four days.

The stiffness of the jaw remains a considerable length of time after the subsidence of the symptoms in other portions of the body.

Meltzer, some years ago, suggested the injection into the spinal canal of a 25 per cent. solution of magnesium sulphate, 1 mil (16 *m.*) of this solution being injected for every twenty-five pounds (11.33 Kg.) of weight of the patient. The writer has used this, and while it will certainly control the spasm, in the majority of cases this appeared to him as a rather dangerous method, as some of the patients have died from respiratory failure, apparently the result of the injection. Even in developed tetanus it is wise to excise the wound, and to use the open method of treatment of the consequent wound.

GLANDERS.

Glanders is a communicable disease, due to the *Bacillus mallei*, and contracted by man from horses. If the disease affects the nostrils it is called glanders, and if it affects the skin it is termed farcy.

Glanders has been frequently contracted in the laboratory by workers using the *Bacillus mallei* for experimental purposes. It exists in man in the acute and chronic form. At the height of the infection, there is redness, swelling of the affected areas, and lymphangitis. There is malaise, followed by fever, and the individual becomes acutely ill, as he does of any other acute infection. Shortly after the infection, the nose becomes involved, the granulomata break down, and there is a discharge from the nostrils. According to Osler, generalized papules occur and have been mistaken for smallpox. The lymph-glands are enlarged, and pneumonia may supervene. These cases are practically all fatal. The diagnosis can only be made certain by cultivation of the causative bacillus from the lesions.

Chronic glanders is a variety of the disease which masquerades in the form of a chronic coryza. Chronic osteomyelitis has been described as the result of infection by the *Bacillus mallei*, also glandular nodules may occur in almost any of the internal tissues.

The diagnosis of these cases may sometimes be made by the use of mallein used in the same manner as tuberculin is used for diagnostic purposes, after the method of von Pirquet.

TREATMENT.

When a case of glanders is diagnosed, the most important measure is to disinfect all the discharges from the nose or from any of the suppurating areas. There is no specific treatment. The treatment must be based on general lines, such as rest, elimination by administration of abundance of water, by resort to the use of digitalis if the heart fails, and by an abundance of fresh air and good food.

Acute forms of glanders are almost always fatal, and many of the cases of chronic glanders end in death.

ANTHRAX.

Anthrax is an infectious disease of domestic animals, communicable to man by inoculation, inhalation, or ingestion of the specific bacterium of the disorder, the *Bacillus anthracis* found in the blood, tissues, and local lesions of infected subjects.

Synonymous terms for the disease are referable in certain instances to the characteristic external signs of the infection (malignant pustule; malignant edema; erysipelatous anthrax); in others to prominent clinical features, such as splenic enlargement and pyrexia (splenic fever); and in still others to occupational predisposition (wool-sorters' disease).

Human anthrax is confined almost exclusively to those whose work brings them in close contact with wool and hides, and the vast majority of cases occur in butchers, tanners, wool-sorters, and drovers.

Malignant pustule and malignant edema are the two types of *external anthrax,* acquired by inoculation through an abrasion, scratch, or cut in the skin, and in this common variety of the disease the chief objective symptoms relate to a small red papule with an inflamed indurated base, rapidly developing into a necrotic pustular mass attended by violent inflammatory edema and considerable adenitis of the neighboring lymph-glands. Exceptionally, a rapidly spreading circumscribed edema, with no definite pustule, constitutes the initial lesion, in the event of which death from overwhelming sepsis is to be anticipated.

Two forms, the pulmonary and the gastro-intestinal, are included in the relatively less common variety of *internal anthrax,* resulting from inhalation of the germs or from eating the flesh of infected animals. Both of these varieties of anthrax are dominated by the constitutional symptoms of septicemia, with a clinical picture of either acute diffuse bronchitis or bronchopneumonia in the first, and with evidences of violent gastro-intestinal disturbance in the latter. These types of internal anthrax carry an unfavorable prognosis, the outlook being particularly bad in gastro-intestinal cases.

External anthrax offers no great difficulty in diagnosis, and the characteristic appearance of the local lesion charged with

the specific bacillus is proof positive of the nature of the affection. In the internal variety, however, the problem is harder to solve, and, while the patient's occupation may afford a clue, such evidence is largely intangible, and should be corroborated by the discovery of the anthrax bacillus in the blood-stream.

TREATMENT.

It is hardly necessary to state that extirpation and thorough cauterization of the local lesion is to be done without delay in every case of malignant pustule. This is accomplished most effectually by the actual cautery, for excision with the knife is more than likely to leave unremoved bits of infected tissue and, furthermore, may disseminate the bacteria through the circulation. If the knife be used, it should be supplemented by the application of the hot iron, carbolic acid, or some similar corrosive liquid, in order to insure a clean and aseptic wound at the site of the primary sore.

Hypodermic injections of a $\frac{1}{20}$ aqueous-etheral solution of carbolic acid under and around the wound and the application of a compress wet with the same solution is advised by Bell. Subsequently the local treatment consists of cleansing with a $\frac{1}{2000}$ mercuric chlorid solution and the application of tincture of iodin to the lesion and to the surrounding zone of inflammation. In favorable cases this routine carried out for a fortnight or three weeks results in healing of the ulcerated focus, with but slight scarring, considering the nature of the infection and the necrosis thereby caused.

In addition to the foregoing methods of caring for the local lesion, all types of anthrax should undergo treatment with Sclava's antianthrax serum, the routine use of which in Italy has within recent years reduced the mortality from this disease from 24.1 per cent., to 6.1 per cent., the present figure (Cavaille). By preference the immune serum is given intravenously, in initial doses of from 80 to 100 mils (21½ to 27 f3), although some prefer the intramuscular technic. Favorable results also have attended the use of stock vaccines made of the *Bacillus pyocyaneus,* and of salvarsan in 0.3 Gm. (4.6 gr.) intravenous injections.

The management of the coincident septicemia, the dominant clinical feature of many cases of anthrax, differs in no

essential from that of other grave septic disorders, the treatment of which is considered elsewhere, and therefore does not warrant further discussion in this place. (See p. 48.)

Finally, the suppression and spread of anthrax demands close attention, and the details of such preventive measures have to do with the rigid inspection and quarantine of live stock; the disinfection of hides, wool, hair, hoofs, horns, bones, and glue stock, and the similar care of vehicles, yards, and abattoirs; and the sanitary regulations observed in slaughtering, relating not alone to this process, but also to the butchers and fellow-workmen. For an interesting account of the prophylactic measures, enforced by this and other countries, the reader is referred to the United States Government publication on the subject. (Bulletin of the U. S. Bureau of Labor Statistics: "Anthrax as an Occupational Disease," Washington, 1917.)

WHOOPING-COUGH.

The bacillus described by Bordet and Gengou is probably the cause of whooping-cough. The certainty of this being the fact is of great importance, both from a diagnostic and therapeutic standpoint, because, as will be seen, suspensions of these live bacilli may be used for diagnostic purposes, and the killed bacilli or vaccine for treatment. If they are not the cause, both the treatment and diagnosis by use of suspension vaccines are, of course, based on wrong premises. In the present state of our knowledge they may be considered as the active cause of the disease.

The disease is transmissible, probably directly from one individual to another, inasmuch as the infected individual is able to disseminate the contagion several feet. One writer says 5 feet is the distance it can be communicated. It is probable that the bacilli, when deposited in any place where they are not killed by air and sunlight, survive for a period of time, and become the immediate source of transmission.

The prophylaxis, therefore, consists of keeping infected individuals (children) out of school, and from public gatherings, and attention to the destruction of all material they expectorate or vomit. The habit of allowing children to

expectorate on the street, and to permit them to expel this expectoration with the vomit on the street, is pernicious.

If children suffering from the disease are taken into a train, or go in the street, there should be taken along some sort of receptacle in which this mucus can be collected and destroyed.

Vaccination against whooping-cough has been tried with some success. Children who are exposed to whooping-cough should be regularly vaccinated with a good stock vaccine.

The symptoms of the disease are well known. There is at first a catarrhal stage, in which the patient appears to have an ordinary bronchitis, but even in this stage there is a tendency for the cough to be rather paroxysmal, and to occur more frequently at night. In a week or ten days the cough becomes distinctly paroxysmal, the "spells" coming on at intervals varying from fifteen minutes to two or three hours. As a rule, the paroxysms are worse at night, and when the individual is excited or cries. The paroxysm is characterized by several short expiratory coughs, followed by a long-drawn inspiration of high-pitched character—the so-called "whoop." Sometimes the paroxysm is accompanied by so much embarrassment of breathing that there is marked cyanosis, and occasionally bleeding from the nose and petechia under the skin. In very severe paroxysms the child may go into convulsions, or an attack of syncope may occur. A paroxysm is very rarely fatal, except in young infants. When death occurs in whooping-cough it is usually the result of one of the complications, pneumonia being the most frequent cause of death.

The diagnosis during the height of the disease is not difficult, the paroxysmal attacks being present in few other conditions. Care must be taken, however, that a mediastinal growth, such as enlarged bronchial glands, is not mistaken for whooping-cough. This condition lacks the history of a catarrhal stage, lasts much longer, and dullness under the sternum and along the vertebral column can be made out by careful physical examination. Cases of tetany might also be mistaken for whooping-cough. In the early stages of whooping-cough, examination of the blood is of the greatest value. There is a leucocytosis often of high degree. The author has seen one of 32,000, with a predominant increase of lymphocytes. An agglutination test also is said to be of value, the

blood of the patient agglutinating the fresh culture of Bordet's bacillus exactly as the blood of a typhoid patient agglutinates the culture of typhoid bacillus.

TREATMENT.

The patient should be kept constantly in the fresh air, either in rooms with the windows open or out-of-doors, depending upon the condition of the weather. On clear days the child should sleep out-of-doors, and when in the room the windows should be widely open. Violent exercise, crying, and laughing are apt to precipitate a paroxysm, hence the patient should not take part in rough games, and should be as free from excitement as possible.

Young infants should be carefully watched. In these and in other frail individuals there is danger from the paroxysm itself, and care should be taken to protect the patient by lifting it in the arms, supporting the head, and otherwise giving support to its body muscles and chest. Kilmer's croup bandage is said to be of some value, particularly in young children. Rurah describes it as follows:

"A stockinette band, similar to those used under plaster jackets, is applied to the body from the axillæ to the pubes, and two shoulder straps are used to keep it from slipping. On this stockinette a width of silk elastic is sewed, so that it goes around the abdomen, and entirely covers it. It should be pinned slightly on the stretch, and then sewed on, so as to keep it from curling."

The protected finger may be used to remove the mucus from the fauces, which often seems to impede the respiration; this use of the nurse's hand also seems to be of value, because the jaw is pushed down, and it should also be pushed forward.

Vaccines. The treatment of whooping-cough by vaccines of the Bordet bacillus is on trial. In the few cases the writer has used it, it has not been of signal value, but favorable reports have been made as to the efficacy of vaccine therapy in the condition, and it is a wise thing to use a stock vaccine of reliable make in every case of whooping-cough.

The various powders and whooping-cough cures which are in the market are mentioned only to be condemned. Various local sprays have been tried, but their varied character and

number prove that none of them are specific, just as the same holds for internal medication.

Food. The frequent vomiting inseparable from whooping-cough often causes much emaciation. To overcome this lack of food it is important to give small amounts of food frequently, rather than large amounts at one time. The diet should consist largely of milk. If the food is vomited, try to persuade the child to eat immediately after the vomiting, for in this way nourishment can be kept up. Of all things, the stomach must be protected from overdosing by drugs.

Internal Medicines. The one drug which has seemed of some value to the writer is belladonna.

This may be given in the form of a tincture. The best method is to begin with 3 to 5 drops (0.2 to 0.3 mil) of the tincture every three hours, increasing the dose 1 drop daily; that is on the first day give 5 drops (0.3 mil) in water every three hours, on the second 6 drops (0.4 mil) every three hours, and so increase until the child gets a physiologic effect, as evidenced by flushing of the face and dilatation of the pupils. The dose should be kept at that point or increased later.

The next most useful drug is bromid of potassium. In many cases full doses of this most useful remedy, 5 grains (0.32 Gm.), every three hours for a child 3 years old, will lessen both the number and the severity of the paroxysms. Care must be taken in the use of large doses of this drug that the little patient is not too badly narcotized. The writer has seen children badly poisoned with the dose recommended, but nevertheless, the drug is of so much value that it should be used, always with due care.

Chloral may be added to the mixture of bromid of potassium, in doses of 2 to 3 grains (0.13 to 0.19 Gm.).

Bromoform and antipyrin have both been highly recommended, but there is a distinct danger in both of these remedies. The writer has rarely used them.

If there is much bronchitis, both chlorid of ammonia and citrate of potassium can be added to the bromid mixture with great benefit.

When pneumonia develops, it should be treated with just as much detail as it is treated under any other conditions.

Frequently the paroxysms are so severe that there is distinct dilatation of the heart. In these cases, digitalis used in full enough doses to tone up the heart muscle is of distinct value. After the attack is over, many of the children are weak and anemic. Attention to their general health, and, above all, fresh air and good food is important. If they are anemic, iron in some form is of distinct value.

Tuberculosis is a frequent sequel, probably as an exacerbation of an old lesion. Great care must be taken to detect this lesion, and not to comfort one's self with the futile thought that the condition is only weakness following the attack.

ROCKY MOUNTAIN SPOTTED FEVER.

This disease occurs in the valleys of the Rocky Mountains in Idaho and in Montana; it has been found also in the valleys of Nevada and Wyoming.

The cause of the disease is not known, but it has been proved by Ricketts and King that it is spread by means of a tick, the *Dermacentor occidentalis*. These authors have transmitted the disease to animals by means of bites of these ticks; they also found that the ova and young of the infected ticks contain the infective material. "A certain percentage of the female ticks, which have acquired the disease as a consequence of feeding on animals, the latter having been infected by other ticks, transmit the disease to their offspring through the eggs. The new generation, during the process of feeding, transfer the virus to certain of the susceptible small wild animals (ground squirrel, rock squirrel, chipmunks, ground hogs, and, perhaps, others), and this may take place either during the larval, nymphal or adult stage; hence at various times of the year. During the infection of the wild animals it is required that hitherto normal ticks, either as larvæ, nymphs or adults, acquire the disease by feeding simultaneously with, or shortly after, the feeding of the infected ticks.

"There is a short period of malaise, followed usually by a well-marked chill. These chills may be repeated throughout the course of the attack. At the beginning there is severe aching of the bones and muscles, pains in the joints, and

severe headache. Constipation is the rule. The skin is dry; the tongue is coated and sordes appear early, and the case has the appearance, except for the chill, of a typhoid in its beginning. The temperature rather rapidly develops, and soon reaches 102° or 103° F. (38.8° or 39.4° C.) on the third or fourth day. It may go much higher, reaching even 107° F. (41.6° C.). There is usually a slight evening increase and morning decrease. When recovery occurs, the temperature falls by lysis.

"The most characteristic part of the disease is seen upon the *skin*. From the second to the fifth day after the chill a macular rash appears around the ankles and upon the wrists, and then extends over the entire body, sometimes in twelve hours. More usually, however, it takes a longer time. A desquamation, best seen on the soles of the feet and the palms of the hands, occurs during the second week of the disease. Occasionally there is jaundice. In severe cases the patients become delirious, and pass into a typhoid state. However, there is no sign of meningitis. The pulse is rapid, and out of proportion to the temperature. The blood is only slightly changed, the erythrocytes being normal, and the leucocytes ranging from 12,000 to 13,000. Except for constipation, there is no unusual sign of disturbance of the digestion. The urine frequently shows the signs of a severe febrile condition, albumin and casts.

TREATMENT.

"The treatment is entirely symptomatic. However, the prophylaxis is an important part. Persons whose business calls them into these valleys in the spring time should be warned of a possibility of infection, and should protect themselves against the bite of ticks in every possible manner, particularly by protecting the feet and hands. General principles should govern the treatment. The patient should be in a cool, well-ventilated room; the diet should be soft and properly regulated. Cold sponging for the fever, with morphin or other opiate, if the patient becomes extremely restless. As a tonic afterward, the patient may be given nux vomica and gentian."

EPIDEMIC PAROTITIS (Mumps).

Epidemic parotitis is a transmissible disease which is highly contagious.

The cause of the contagion is not known. The disease is characterized by a period of incubation lasting from one week to two weeks, and by a period of invasion of shorter duration, one or two days, in which the temperature rises to 100° or 101° F. (37.7° or 38.3° C.). Then the parotid gland on one side gradually enlarges in front of and underneath the ear, pushing the lobe of the ear forward, and extending backward under the sternomastoid muscle. In from two to three days the gland on the opposite side enlarges, and then the child presents the picture of a swelling surrounding his neck, composed of enlarged glands and indurated tissue. Other salivary glands may also become enlarged and painful. Sometimes the submaxillary and sublingual glands are the first to become affected, though this is not the rule. The lachrymal gland also may become affected. There are cases on record in which the symptoms have resembled those of an acute pancreatitis, indicating that the pancreas has taken part in this disease.

During the height of the disease the temperature rises to 102° or even to 104° F. (38.8° to 40° C.), but remaining at this height for only a short time. At the end of a week the temperature subsides, and the individual becomes convalescent.

The disease as seen in the ordinary practice of medicine is of very slight importance, but when it occurs in communities, such as soldiers in barracks or children in orphan homes, it is a more serious disease, and many fatal cases are reported. In these fatal cases there often is a typhoid state, resembling typhoid fever, or nervous symptoms may supervene, delirium, and even convulsions.

Suppuration of the gland is very rare, gangrene of the gland has been reported. After the subsidence of the swelling in the gland, and the child has become convalescent, one or the other testicle is apt to be affected. The testicle becomes hard, swollen and painful, and remains so for several days. The cause of this orchitis is not known; it has been suggested, however, that it is due to an antogenous infection,

the individual transferring the virus by means of the hands to the penis, and from there the infection travels to the testicles.

In females the breasts sometimes enlarge, and there are cases on record where the ovary has become affected.

TREATMENT.

Every case of mumps should be kept in bed during the height of the disease. This particularly applies to males, because it appears that those individuals who are up and about during or shortly after an attack are very much more likely to develop an orchitis; this orchitis being of more or less serious import, atrophy of the testicles occurring rather rapidly, and when both testicles are affected, of course, this atrophy presents the serious sequela of inability to procreate.

The pain in the salivary glands may be relieved by hot fomentations, by hot poultices, by a hot solution of saturated magnesium sulphate. On the other hand, cold, to certain individuals, is more grateful than heat. If fever is very high, the use of citrate of potassium, and sweet spirits of nitre, 10 grains (0.6 Gm.) of the former, to 15 drops (1 mil) of the latter, sometimes appears to do good. If the fever continues high, cold sponging may be used to allay this symptom. The bowels should be kept moved by a mild laxative, and a soft diet should be advised. For the orchitis, applications of heat or cold, as the case may be, and use of a suspensory of some sort while the testicle is swollen, will give a great deal of comfort.

If there is much depression, if the individual is suffering from the toxemia, a stimulant should be used in the form of ammonia, caffein, and strychnin. Large amounts of water should be given to combat the toxemia.

ACUTE POLIOMYELITIS (Infantile Paralysis).

This is an acute communicable disease, due to a virus located in the central nervous system, and in the discharges from the nose, throat and intestine. Colmer, of Long Island, suggested in 1841 that poliomyelitis is a communicable dis-

ease; and Wickman, of Stockholm, proved this to be true by clinical methods and careful observation.

It remained for the laboratories to put the question of communicability beyond doubt, by the fact that they communicated the disease to monkeys by injecting into them portions of the spinal cord and brain of human beings dead of the disease. Later Flexner and Noguchi isolated the germ of this infection and cultivated it.

In the sick, the virus is located in the central nervous system, and in the discharges from the nose, throat and intestine. In the well, the virus unfortunately cannot be identified by ordinary laboratory methods, and therefore we lack a laboratory diagnosis in the so-called abortive cases, such as we have in diphtheria and typhoid fever.

Wickman described eight types of this disease:

1. The spinal poliomyelitis type.
2. The form resembling Landry's paralysis.
3. The bulbar or pontone type.
4. The encephalitic.
5. The ataxic.
6. The polyneuritic.
7. The meningitic.
8. The abortive.

From the standpoint of prophylaxis and treatment, the abortive type is by all odds the most important. The diagnosis is difficult, as will be shown later, but a probable diagnosis can be made in practically all cases. In a suspected case every care should be taken that a diagnosis is made when possible. For practical purposes, the important types are:

1. The abortive type.
2. The meningitic type..
3. The common paralytic type.

As yet the virus has not been discovered in the blood of those sick with the disease, although in one case a bedbug which had fed upon the blood of an infected monkey was shown to harbor the virus, but these bedbugs did not convey the disease to other monkeys by biting them. The virus enters the healthy human being probably always through the nose and throat. The virus resists the greatest heat of summer, and it also resists drying. It may be modified by daylight, and it may be easily destroyed by bright sunlight.

The foregoing facts being admitted, there can be no question but that the disease should be regarded as a specific infection affecting the gray substance of the spinal cord. Yet, inasmuch as nervous symptoms dominate the clinical picture, it seems wiser to consider in detail this type of poliomyelitis in connection with nervous diseases, just as the symptoms-complex of tabes and paresis are discussed under this group of affections, rather than with luetic infections. The reader is, therefore, referred elsewhere for a full consideration of the various phases of acute poliomyelitis, its treatment, and general management. (See section on "Nervous Diseases," p. 670.)

RHEUMATIC FEVER.

Rheumatic fever is an acute infectious disease, characterized by fever, drenching sweats, multiple arthritis, anemia, and a tendency toward inflammation of the endocardium, with complete recovery of the joint condition.

Poynton and Payne have described a micro-organism, the *Micrococcus rheumaticus,* which they believe is the specific cause of this acute disease. This observation, however, has not been fully confirmed by all laboratory workers. Because of the loose way the name of rheumatism is given to joint affections, it will perhaps be wise to attempt briefly to differentiate rheumatic fever from other conditions causing arthritis.

The disease usually begins suddenly, often with a chill followed by high fever, but occasionally the onset is insidious, being preceded for a longer or shorter time by general ill health, by anemia, and other signs of weakness. In the beginning there is often sore throat, aching in the limbs, and slight fever before the acute symptoms occur. The arthritis is one of the most characteristic symptoms of the disease. It is multiple; one joint, usually one of the larger joints, the wrist, elbow, or knee, becomes painful, swollen and red, and liquid develops in or around the joint. The pain in these inflamed joints is extreme, and the individual cries out with pain on the least attempt at voluntary movement, or, indeed, when the joint is moved by the nurse. Frequently several

joints are affected at one time. Sometimes one joint is inflamed, becomes relatively better, then a second joint is implicated; this becomes better, and the third joint is affected, and so on, until practically every joint in the body may suffer. An anemia rapidly develops, and a leucocytosis is usually present. Severe drenching sweats at the height of the fever are very common. The pain in the swollen and inflamed joints is often so great that the patient voluntarily immobilizes himself, and complains bitterly, even when the bed is moved. The temperature range is high, 102° and 104° F. (38.9° to 40° C.), and it may rise even in an ordinary case to 105° F. (40.6° C.).

The complications of the disease are extremely important. Of the greatest importance is inflammation of a heart valve. An endocarditis develops in a large majority of the cases of true rheumatic fever. The valve most frequently affected is the mitral, although an aortic valve may be implicated. Pericarditis may occur, as well as endocarditis, either independently or as a part of the serous membrane infection. This pericardial inflammation is characterized sometimes only by an audible friction murmur; at other times there is pain in the region of the heart, sometimes so severe that it may be transmitted to the abdomen, and mistaken for appendicitis. Occasionally the physical signs appear most rapidly, within an hour from the time a careful examination has been made, and no friction rub or pain elicited, and a second examination may show a loud friction murmur. If an effusion occurs, the murmur may disappear as quickly as it appears.

Hyperpyrexia. The patient may become wildly delirious, even maniacal, and the temperature rise to 105° or 106° F. (40.6° or 41.1° C.); he may die in this state from excessive fever.

Where the infection is very severe, the endocarditis and pericarditis may be accompanied by an actual pneumonia and pleuritis. Pulmonary complications may also occur alone.

Of the nervous symptoms, delirium is perhaps the most common. Chorea occurs probably as part of the manifestation of the rheumatic infection in the spinal cord and brain, and in the meninges.

One often sees this combination: first, a slight attack of chorea, which may advance to quite a severe attack, and this is either preceded or accompanied by tonsillitis; then the curious fibrous nodules occur; finally, an endocarditis. The writer has notes of such a case, in which tonsillitis, chorea, fibrous nodules, a slight arthritis, and a very severe endocarditis occurred, the child losing her life in the course of three or four years from the endocardial infection.

Notwithstanding the occurrence of such cases as these, the exact relationship of rheumatism and chorea is still not accurately established.

Cerebral Rheumatism. This name is given to a condition characterized by wild delirium, coma, and sometimes convulsions and hyperpyrexia. Osler states that in certain cases of this sort seen by him he considers the salicylates which had been given were the cause of the untoward symptoms. This so-called cerebral rheumatism may occur in the midst of the disease, or in the very beginning.

Rheumatic nodules are small bodies which occur along the sheaths of the tendons, usually about the elbows, hands and wrists. They are painless, movable, and, according to Payne, are truly inflammatory objects, and are not composed of fibrous tissue.

Pneumonia and pleurisy both occur. There is nothing peculiar in the symptoms of these conditions when they complicate rheumatism, but probably when they occur during the attack of rheumatism are part and parcel of this infection. They are most commonly associated with the cases which also have endocarditis as a complication.

Erythema, frequently in the circinate form, commonly develops during an attack of rheumatism.

Erythema nodosa is characterized by nodular infiltration, usually upon the calves and shins, the nodules being raised, dark red or purplish, and extremely painful. The exact relation of erythema nodosa to true rheumatism is not known.

Mild Forms of Rheumatism. Perhaps as important as any other rheumatic infection are the very mild forms, where the arthritis is extremely slight, and often a symptom which does not call the attention of the patient to his condition. The so-called "growing pains" of the laity are unquestionably, in

certain instances, true rheumatism, as proved by the fact that these so-called "growing pains" are very frequently accompanied by endocarditis and other affections, which frequently complicate true rheumatism; therefore, when a child complains of vague pains in his joints, careful search should be made of the throat, of the heart, and of the extremities, to be sure that one is not dealing with a true rheumatic infection, and also to be sure that he is not overlooking the endocarditis so often incident to this condition.

As stated in the beginning of this section, the differentiation of rheumatism from other arthritic conditions is so badly done, except where careful study of the case is made, that perhaps it will be wise to take into consideration very briefly the differentiation of these various conditions.

First, to deal with so-called muscular rheumatism. Almost without exception, muscular rheumatism is a form of myalgia due to infections other than rheumatism. Rarely are these muscular affections accompanied by any of the symptoms or complications common in true rheumatism. There is no arthritis, no endocarditis, and often no fever.

Multiple Secondary Arthritis. Secondary arthritis occurs as a result of many infections, such as gonorrhea and syphilis, and is often the result of a toxin elaborated by the poisons of scarlet fever and diphtheria. The great and important point in the diagnosis and differentiation of these conditions from rheumatism is to search for the causative factor. Too frequently a polyarthritic condition of gonorrheal origin is treated as rheumatism, until the joints are deformed, and until much harm has been done. If the case had been carefully studied, and the cause, gonorrhea, found, doubtless the individual would have gotten well without deformed joints. Rheumatism is often mistaken for a traumatism when it chances to be localized to one joint, especially the knee-joint, or in one of the extremities. Of course, the differentiation here depends entirely upon the history as to whether the individual has, or has not, suffered an injury.

Arthritis deformans is frequently mistaken for rheumatism. In this condition, in the ordinary form, the individual develops pain, stiffness, and deformity of various joints, often and usually either the joints in the hands or the feet. When

the condition is acute, it resembles in many particulars acute rheumatic fever, but the joints soon become deformed, are fixed, and remain in that condition for months, and perhaps habitually. This is an entirely different picture from the acute onset or acute condition of the joints in rheumatic fever.

The harm which has been done in these cases of rheumatoid arthritis or arthritis deformans is that the focus of infection, which is the cause of these destructive arthritic conditions, has not been searched for, and the patient becomes a chronic invalid before the investigation is made. Again, the arthritis deformans patient is put on all sorts of diet entirely unsuited to his condition.

Gout is a condition which may be mistaken for rheumatism. Usually, in the typical cases, gout is sudden in its onset, affecting by predilection the second joint of the great toe; there is sudden fever, sudden swelling and redness of the joint, and the patient is extremely ill for two, three, or four days, and then rather abruptly becomes convalescent, with a rapid disappearance of the arthritic signs. In chronic gout there is, almost without exception, a deposit of the biurate of soda in and about the joint, and these deposits may be found upon many of the cartilaginous parts, such as the pinna of the ear. This, again, is a condition which is entirely different from true rheumatism.

Epiphysitis, of septic origin, is unfortunately frequently mistaken for rheumatism. When the epiphyseal extremities of a joint are affected in a child, and particularly when only one joint is affected, one's suspicion should always be aroused. The affected part should be carefully *x*-rayed, put at rest at once, and immediate surgical interference made, when the condition seems to be in the bone, and not in the joint itself. Many children have lost their lives by considering this inflammation of the bone itself a true rheumatism, until destruction of the bone has occurred.

Osteomyelitis, another condition, affecting other portions of the bone, is also mistaken for rheumatism. The author has seen deaths from it, the result of treating it as rheumatism for weeks, and finally necessitating serious operation.

In all these cases, if the observer remembers that true rheumatism is an acute infection, beginning suddenly, as

many other acute infections do, often with sore throat, high fever, anemia and leucocytosis, and that the joints are affected early, usually as a multiple arthritis, almost without exception one can come to a quick decision, and differentiate the arthritic conditions of which I have spoken from rheumatism without much trouble.

Of course, occasions arise in some cases where one is in grave doubt as to the true cause of the joint trouble. When one is in such doubt, however, he should be careful to give the advantage toward the more serious condition.

TREATMENT.

The lightest case of rheumatic fever should be at rest in bed, for the reason that not only may the joint condition be limited by the rest, but that, even in the mildest cases, endocarditis is likely to occur, and when this takes place it can be treated properly only by the insistence on complete rest from the very beginning of the illness.

When practicable, the affected joint should be fixed in some sort of a splint, in order thus to prevent movement, and possibly to limit the inflammation, and also to relieve the pain from which the patient suffers. Applications to the joints of ointment of menthyl salicylate, or of a saturated solution of magnesium sulphate, or some warm application, such as an electric pad or hot-water bag, give comfort to the patient.

If the joint is surrounded by large pads of cotton, this alone sometimes seems to afford relief.

When a joint is fixed on a splint, it must be held in a position which is comfortable to the patient. Simply to put a posterior splint upon a leg where the knee is affected is often to give extreme pain to the patient.

If, on the other hand, the joint be fixed in the position which it assumes because of the swelling of the joint, the patient will be comfortable. If the case is one of true rheumatic fever, there is no danger of the joint becoming permanently fixed in an abnormal position, because, as stated in the beginning of this article, the arthritis entirely recovers in true rheumatism, and, indeed, this entire recovery of an arthritis is a mark of the true rheumatic character of the condition.

When the patient is bathed in sweat, this annoying symptom can be greatly relieved by carefully sponging the body with tepid water, and then sponging off lightly with alcohol. This gives a great deal of comfort to the patient, who is annoyed with this extremely unpleasant symptom.

As intimated above, the portal of entry in this condition is frequently the tonsils. The tonsils therefore should be carefully watched, and applications of an ordinary Dobell's solution, boric acid solution or simple normal salt solution, should be made, preferably in the form of sprays. It is the custom of certain physicians to recommend that inflamed, infected tonsils should be removed in the course of the acute infection of rheumatism. To the writer's mind, this is an error. He has seen so many serious complications result from the removal of the tonsils during the acute infection, that he feels that it is a dangerous procedure. However, when an individual has had a number of attacks of tonsillitis, accompanied by rheumatism, and the tonsils remain cryptic during the intermissions, these tonsils certainly should be removed.

The hyperpyrexia and the cerebral symptoms which accompany rheumatism, are best treated by cold sponges or cold baths, and in cases where the delirium is extremely severe, by blood-letting.

The pain, which is extremely severe in certain instances, should be controlled by morphin, when it is severe enough to prevent the patient from resting; rest, as said before, being the essential point of the treatment. Care, however, must be taken, when morphin is given under these conditions, that the administration be carefully controlled, in order that the patient be not allowed to develop a morphin habit.

Care of the teeth in the very early stages of rheumatism is of the greatest importance. Sordes about the teeth and old pyorrhea will often keep up indefinitely the attack of arthritis, which is apparently a true rheumatism. In such instances cleansing of the mouth will cause a cessation of the attack.

Internal Medication. Some compound of salicylic acid is used the world over in every case of acute rheumatism. It seems to the author that the administration of this drug is of the highest importance. In the first place, when a case is

proved to be a case of rheumatic fever, it should be given one of the salicylates in large enough and frequent enough doses to bring about the therapeutic symptoms of the administration of that drug. Salicylate of soda can be given in 15-grain (1 Gm.) doses, well diluted, every three hours, until the subject complains of tinnitus and nausea, or of one of the other untoward symptoms of the administration of the drug. When it brings about gastric disturbance it may be combined with bicarbonate of soda in equal amounts. This routine should be kept up for four or five or six days during the attack of the rheumatism. If, in the course of five or six days, or perhaps a week, no good results have come from the administration of the salicylate in this form, then either the form of the drug should be changed or the salicylate administration should be stopped entirely. I am quite sure that I have seen many cases of deranged digestion, depression, and weak heart from a very prolonged administration of the salicylates, in cases where they were doing no good. Other forms of salicylates may be used. Acetyl salicylic acid (aspirin), given in 10-grain (0.66 Gm.) doses, every three hours, is often more beneficial and gives more relief than does the salicylate of soda. Phenol salicylate or salol may be tried, but in my hands it has not been nearly so efficacious as either of the other two.

Salicylic acid, in doses of 10 grains (0.66 Gm.), given in capsules every three hours, of course may be tried, but it always seems that the administration of the acid causes more digestive disturbance than does the use of one of its salts. The administration of alkalies often gives relief; these may be used in the form of citrate of potassium, 15 grains (1 Gm.) every three hours; or as bicarbonate of soda, combined with bromid of potassium, 10 grains (0.66 Gm.), every three hours. If the joints do not very rapidly get better, if after all acute symptoms have disappeared, they still remain swollen and stiff, very light massage may be guardedly practised. Great care, however, must be taken that this massage does not give pain to the patient. While in suitably selected cases this procedure may be of great value, on the other hand, it may do the greatest amount of harm; and I am sure reinfection occurs by massaging the joints too early. Therefore it is

better to err on the side of safety, and to allow a joint to be untouched if it gives the slightest amount of pain to move it. Certainly indiscriminate rubbing by members of the family should be severely frowned upon.

If the arthritides resolve slowly, light blisters over the joints, the blister being allowed to remain one or two hours, or slight applications of the actual cautery, often will start a resorption of the exudative material. Baking with superheated air is of value.

It has been stated above that endocarditis is the one complication of rheumatic fever which is of the greatest importance. Therefore daily examination of the heart of rheumatic subjects should be made throughout the entire course of the disease, and the slightest appearance of any endocardial trouble should be a sign that the patient must remain quiet until all fever, leucocytosis, and local signs of cardiac disease have disappeared, except, of course, the murmur, which tends to remain permanently.

The question as to when the patient shall get out of bed is always one of great importance. Usually as soon as the acute attack has disappeared, the patient remains still slightly stiff, with a small amount of pain, but he clamors to get out of bed. He should be kept flat on his back until absolutely all of the acute symptoms have disappeared, particularly until the joints can be freely moved without pain, and until the fever and the leucocytosis disappear permanently. Too early rising is the reason for prolonged attacks of rheumatic fever which have relapse after relapse.

The management of convalescence is important. The patient is weak and anemic, and therefore should be well clothed and kept at rest in the open air, allowing him to move about only a small amount at first, and gradually increasing the exercise as he becomes more convalescent. He should be given an abundance of good food—meat, milk and eggs—a mixed general diet. The old habit of forbidding cases of acute rheumatic fever from the use of meat is a mistake.

Medicines for the convalescent are important. Where the joints remain stiff, the use of iodid of potassium, 5 grains (0.34 Gm.), three times a day, is perhaps of some importance, but if the administration of this drug causes a loss of appetite

it is better to discontinue its use. Iron is of value; iron in the form of Blaud's pills, 5 grains (0.34 Gm.), three times a day, care being taken that the pill is one of the soft variety, or that the pill mass is powdered and taken in capsules. Basham's mixture can be used in lieu of Blaud's pills, and is an efficacious method of administering the iron. Where the anemia is very severe, hypodermic injections of the citrate or lactate of iron can be used, instead of administering this metal by the mouth. Some authors believe this is the only proper way to administer iron, but with this opinion the author does not agree, as he has seen rapid convalescence and rapid blood reconstruction occur under the use of Blaud's pills.

The Use of Vaccines. So far as the author is aware there is no well-founded proof that the use of rheumatic vaccines is of the least value in the treatment of acute rheumatism. Certainly the use of stock vaccines is not justifiable. If Poynton's and Payne's views (that the *Micrococcus rheumaticus* is the cause of the disease) be well founded, then possibly the use of vaccines of this bacterium may be of some value, but thus far it has not been proved to be of any use. There is no serum which is of value. The administration of the various remedies on the market, in the form of rheumatic serums, phylacogens and rheumatic vaccines, is not to be advised. On the other hand, it has been lately shown that the intravenous injection of almost any foreign proteid, in certain cases of arthritis, whether they are rheumatic or not, is followed by symptomatic cure.

It seems, however, to the writer that this method of treating arthritis is not to be undertaken without care. One may surely remove the pain and swelling of certain cases of arthritis by the administration of ¼ minim of Colle's fluid, or of foreign protein of some other form, such as typhoid vaccine, but the question still remains whether one is not simply treating a symptom instead of removing the cause. The symptom surely should be treated, but the case should not be dismissed with their disappearance as the only evidence of cure. The use of foreign proteins in this way also occasionally brings about such severe reactions that the patient's life may be jeopardized.

ACUTE TONSILLITIS.

Acute tonsillitis is an inflammation of the tonsils, characterized by redness of the tonsils, and, indeed, of the whole pharynx, and by an exudate upon the tonsils, frequently occupying the follicles alone, sometimes spreading over the entire tonsil in a pultaceous mass.

The general symptoms are a chilly sensation, aching of the limbs, headache, resembling in every way infectious colds, but with the added inflammation of the tonsils, as stated above.

The micro-organisms, which are usually found in this infection, belong to either the streptococcus or staphylococcus group, the more severe forms, however, being a true streptococcic sore throat. Rarely are these bacteria found in pure culture but pneumococci, micrococci catarrhalis, and many other forms of bacteria are found.

The disease is highly transmissible, and every case of tonsillitis should be isolated, where it is at all practicable. The danger in this particular disease is the possible mistaking diphtheria for this infectious form of sore throat. What has been said in regard to cultures of the throat in diphtheria should be carried out in every case of exudative sore throat, and, when there is any grave reason for doubt that the case is one of diphtheria, a protective dose of diphtheria antitoxin should be given. Another danger of follicular tonsillitis is that the tonsils thus become a portal of entry for other organisms, and eventually arthritis, endocarditis, and other serious conditions arise as secondary deposits of the infecting organism.

TREATMENT.

Every case of follicular tonsillitis should be put to bed, and kept in bed until it is entirely well. Local treatment is often of great value. The use of ice held in the mouth, or of an ice poultice, often gives considerable relief. Sprays of a mild antiseptic solution, such as suggested for infectious colds, that is Dobell's solution, or normal salt solution, are of the greatest value. The application of a 5 per cent. solution of nitrate of silver, of tannic acid in glycerin, 20 grains (1.3 Gms.) to

the ounce (30 mils), and of bicarbonate of soda spread over the tonsil also is advised. A mixture of tincture of chlorid of iron and of bichlorid of mercury in lemon-juice and syrup, is, in the writer's practice, of more actual curative value than any other medication. The tincture of chlorid of iron may be used in 10-drop (0.62 mil) doses, and the bichlorid of mercury in from $\frac{1}{24}$ to $\frac{1}{50}$ of a grain (0.002 to 0.001 Gm.) every three hours. The prescription may be written as follows:

℞ Hydrargyri chloridi corrosivi ..... gr. ¾ (0.048 Gm.).
Tincturæ ferri chloridi,
Succi limoni .....āā f℥ss (15 mils).
Syrupi .....q. s. f℥iij (90 mils).
M. S.: Teaspoonful (4 mils) every 3 hours.

The salicylates, sodium salicylate, in 15-grain (0.97 Gm.) doses every three hours, phenol salicylate (salol) every three hours in from 5- to 10- grain (0.32 to 0.65 Gm.) doses, acid-acetyl-salicylic (aspirin) in 5-grain (0.32 Gm.) doses are of value, but are of less value than the iron mixture given above.

After the attack of tonsillitis, the individual is apt to be very weak, and should avoid overexertion. During the attack and after it, the heart should be very carefully examined, because, as said previously, endocarditis is one of the sequels of these attacks of tonsillitis, and the early treatment of endocarditis is the only treatment which is of actual curative value.

FEBRICULA (Ephemeral Fever).

This title is given by Osler to a set of symptoms characterized by fever, without any other diagnostic symptoms. There are unquestionably many cases where there is fever, in which we are unable to find the cause of infection. It seems to the writer, however, that perhaps the name of infection of unknown cause would be better than to give the name febricula or ephemeral fever to this class of cases. The reason for this is that we are satisfied with the name febricula, and it may tempt us to cease our efforts to find the cause for the outbreak.

TREATMENT.

The rule to follow in these cases of unknown infection, in order to be on the safe side, is to consider the case as some

definitely known infectious disease, such as typhoid fever, measles, scarlet fever, etc., the one it most closely resembles, until the opposite is proven to be the fact. In this way we will avoid treating cases under the name of febricula which are really abortive cases of any of these infectious diseases. The treatment necessarily is entirely symptomatic. Rest in bed, cooling drinks, freedom from excitement and exposure are all that can be done until an actual diagnosis is made.

INFECTIOUS JAUNDICE (Weil's Disease).

This condition is characterized in some cases by gradual onset, and in other cases by sudden onset, of developing jaundice, enlargement of the liver and splenomegaly. Sometimes there are decided nervous symptoms, delirium being very common. There is marked pain in the abdomen.

In the tropics the disease may be mistaken for mild cases of yellow fever or of dengue. It may also be mistaken for simple catarrhal jaundice, or for one of the more grave cases of infectious jaundice, or for acute yellow atrophy of the liver. All these conditions must be taken into consideration in every febrile attack characterized by abdominal pain and by jaundice. The specific organism, if one exists, is not known.

TREATMENT.

The treatment is entirely symptomatic. Rest in bed is essential, and, if the temperature is high, hydrotherapy should be practised. If abdominal pain is severe, hot fomentations are useful. If the toxemia is great, large amounts of water, very mild laxatives, and often the intravenous and subcutaneous use of salt solution is of great value. Severe purgation is contraindicated, because there is always more or less inflammation of the gastro-intestinal mucous membranes, and these purgatives certainly irritate, and increase this inflammation.

There is a great deal of prostration following these cases, and the convalescence is often prolonged. Here the individual should take care not to return to his ordinary vocation for a considerable time after the attack. He should rest in the open air, take large amounts of digestible food, such as milk, eggs and cereals, and as much meat as he can possibly digest with comfort.

GLANDULAR FEVER.

This disease was first described as an entity in 1889 by Pfeiffer. It occurs usually in young children. The patient complains first of sore throat with some redness of the fauces, then the superficial glands become involved, particularly those of the post cervical region. The temperature rises to 101° to 103° F. (38.3° to 39.4° C.); the enlarged glands vary in size, often being as large as an egg. The disease is unquestionably of an infectious character, and occurs commonly between October and June. The glands are tender to the touch, and the liver and spleen are frequently enlarged. Where practicable, the children should be isolated, kept strictly in bed, and, on account of the danger of inflammation of the kidneys, the diet should be light. Sprays to the throat should be used.

West, in an epidemic of 96 cases, has found small doses of calomel to be the best drug to use.

The patients are extremely weak, and require tonics, fresh air, and good food as after treatment.

ACTINOMYCOSIS.

Actinomycosis, a disease rather common in cattle, is sometimes communicated to man.

The writer's knowledge of actinomycosis and glanders is very limited, and derived largely from the literature.

The parasite to which the disease is due is the *Streptothrix actinomyces,* and can be found in the discharges from the lesions of the disease. If it affects the lungs, actinomyces may be found in the sputum. The digestive tract has been affected. The actinomyces have been found in the cavities of the teeth. Frequently the jaw is affected. Enlargement of the jaw on one side may occur, which looks extremely like sarcoma, the diagnosis being made only by the discovery of the actinomyces. The tongue has been infected, small nodules occurring over various parts of the tongue. Actinomyces have been found in the cecum, and in the appendix, the intestinal form being usually regarded as appendicitis. Of course there is no possibility of a diagnosis of the nature of the appendicitis, unless there be some superficial lesion.

Pulmonary actinomycosis is characterized by cough, fever, wasting, and mucopurulent expectoration. The lesions are usually widespread or there may be simply a chronic bronchitis, and here, as stated above, the diagnosis is made by discovering the actinomyces in the sputum.

Miliary actinomycosis is a variety in which the lesions are scattered nodules throughout the lungs, and unless there be some superficial lesion the diagnosis is not possible before death.

There are other cases in which the disease is more destructive, and affects larger portions of the lung. The writer once saw a case which simulated closely a pleural effusion. The chest was tapped; a small amount of liquid was obtained; the lung was then explored, and the material resembling the sarcoma of a pleura was recovered, which disclosed the presence of actinomyces.

Cerebral actinomycosis is described in isolated cases, the diagnosis here depending entirely upon the recovery of the actinomyces from the parts. As Osler says, the disease is in reality a chronic pyemia. Secondary foci may occur both in pyemia and in actinomycosis. The tendency, however, is rather to the production of a local purulent infection which erodes the tissues, and is very destructive.

TREATMENT.

When the disease is local, the focus should be entirely removed, and the cavity painted with iodin. In the generalized infection, the use of iodid of potassium large doses, 40 to 60 grains (2½ to 4 Gm.) has sometimes proved curative. *X*-rays have been used about the superficial lesions with beneficial results. Certainly, however, if a local lesion exists, it had better be excised, curetted, painted with iodin, after which the use of the *x*-ray may be tried in order to kill any germs lingering in the adjacent tissues.

HELMINTHIASIS.

The diseases due to helminths or worms vary with the type and location of the parasite infesting the individual.

These parasites may be divided into those whose habitat is the intestinal canal, and those infesting other portions of the body. Of the intestinal parasites the most common are the flukes, or *trematodes;* the *cestodes,* or tapeworms; and the *nematodes,* or round worms.

The first of these, the flukes, or *trematodes,* have flattened leaf-shaped bodies. Infestation by them gives rise to so-called distomiasis. In these conditions particularly the symptoms depend upon the site of the lodgment of the trematodes. These forms are recognized:—

1. Pulmonary distomiasis.
2. Hepatic distomiasis.
3. Intestinal distomiasis.
4. Haemic distomiasis—bilharziasis.
5. Schistosma Japonica oel Cattoi.

PULMONARY DISTOMIASIS.

This is due to infection by *Paragonimus westermanii.* The symptoms are chronic cough, yellow or red sputum, and the presence of enormous numbers of the ova in the sputum. It is constantly mistaken for tuberculosis, and can be differenitated only from this condition by the examination of the sputum. It is common in Japan and China. According to those familiar with the disease, there is no specific treatment. General measures, disinfection of the sputum, removal from the region in which they become infected, are depended upon to relieve the sufferers.

HEPATIC DISTOMIASIS.

There are a number of flukes (Distoma) found in the liver of the different individuals who are infested. According to Osler, "Six species of liver flukes are known to occur in man. More specifically these are: (1) the common liver fluke (*Fasciola hepatica*), which is a very common parasite in the ruminants. It is a rare and accidental parasite in man, but in Syria a strange disease called 'Halzoun' is caused by eating raw goat liver infected with the parasite. (2) The Lancet fluke (*Dicrocoelium* [*Distoma*] *lanceatum*). (3) *Opisthorchis* (*Distoma*)

felineus, which is found in Prussia and Siberia, and by Ward in cats in Nebraska. (4) *Opisthorchis noverca* (*Distomum conjunctum*), the Indian liver fluke described in man by McConnell. (5) *Opisthorchis* (*Distoma*) *sinensis,* which is by far the most important of the liver flukes, and occurs extensively in Japan, China, and India. It is 10 to 20 mm. long by 2 to 5 mm. broad. The eggs are oval, 27 μ to 30 μ by 15 μ to 17 μ, dark brown with sharply defined operculum. A number of imported cases have been found in Canada and in the United States. White found 18 cases in San Francisco."

The liver is enlarged, cirrhosis occurs, diarrhea develops, at first appearing in attacks, and later being almost continuous. Occasionally jaundice appears. The diagnosis can be made easily, and only, by discovery of the ova in the stools.

The same unsatisfactory treatment must be used—removal from the infected area, and sustaining the patient with general measures. Laparotomy has been suggested in order to reach the bile ducts and remove the parasites from the ducts.

In the infected areas the species of fluke infecting the individual should be known, and then the source of infection destroyed—infected cats and dogs, and avoidance of eating raw liver, when sheep, cattle and goats are the hosts of the fluke.

INTESTINAL DISTOMIASIS.

According to Stiles, several species of flukes are responsible for this condition. The stools should be examined. The symptoms of some are *nil,* in others there are diarrhea and bloody stools.

Thymol and calomel are used to rid the intestines of the parasite.

HEMIC DISTOMIASIS; BILHARZIASIS.

This infection is due, according to Stiles, to two or possibly three species of flukes, the Asiatic and African blood-fluke. In Egypt, epidemic hematuria has been known for a long while, and is one of the most important of parasitic diseases. It is due to infection by the African blood-fluke, described by Bilharz in 1851 or 1852.

According to Stiles, the complete life history of the parasite is unknown, but evidence is accumulating which indicates that the parasites gain access to the human body through the skin, though the embryos may be taken into the intestinal canal through impure water. The ova and parasites are found in various parts of the body, causing irritation, fibroid changes and papillomata of the rectum and bladder.

The symptoms vary with the intensity of the infestation, and with the particular parts most involved. When the seat of the most severe infestation is greatest in the genito-urinary system, "irritability of the bladder, dull pain in the perineum, and hematuria are the commonest symptoms."

Should the rectum be involved, the most prominent symptoms are "bloody stools, diarrhea, prolapse of the rectum, and papilliform growths." In common with many other parasitic affections, there may be severe infestation without any definite symptoms.

The treatment is highly unsatisfactory. No remedy is known which will destroy the ova in the blood. Operative measures upon the bladder and rectum are sometimes performed for the local condition. If perineal fistulæ occur they are treated surgically, as is prolapse of the rectum.

Male fern, santonin, quinin and methylene blue have all been recommended; quinin and methylene blue with the hope of affecting the ova or embryos in the blood; santonin and male fern, of course, only for the purpose of ridding the intestine of the parasite.

DISEASES CAUSED BY CESTODES.

Tapeworm infection is characterized by the presence of the adult worm in the intestine, and the larvæ of the tapeworm in various other organs. The tapeworms which give rise to symptoms and which are of importance in clinical medicine in this country are:

1. *Tænia solium,* or pork tapeworm.
2. *Tænia saginata,* or beef tapeworm.
3. *Dibothriocephalus latus* or fish tapeworm.

The symptoms of these three infestations may be taken up together. First, the presence of any of the tapeworms may

give rise to absolutely no symptoms. All these varieties of tapeworms appear very largely in the United States, indeed, around the entire world. *Dibothriocephalus latus* (fish tapeworm), however, is common along the Baltic Sea, in parts of Switzerland, and in Japan. The beef tapeworm is due to eating uncooked beef which contains the larvæ of the worm, and *Tænia solium* is due to eating uncooked pork, which contains the ova of the pork tapeworm. The *Dibothriocephalus latus* invades the human intestine probably from the eating of uncooked or undercooked fish, bearing the larvæ of this worm. The heads of both the beef tapeworm and the pork tapeworm are extremely small, being a black object about the size of a pin head. The pork tapeworm head is round and has four sucking-disks, and two rows of hooklets around it. By the means of these sucking-disks and the hooklets, the parasite gets its hold on the mucous membrane of the intestine. The length of a mature tapeworm is from six to twelve feet.

The head of the beef tapeworm is rather square, has four sucking-disks, and has no row of hooklets around it. It is by means of these sucking-disks that the head makes fast to the mucous membrane of the intestine. The length of this tapeworm is from fifteen to twenty feet, or more.

The head of the fish tapeworm is different from the other tapeworms, is long and narrow, and has a slit on one side by which it holds fast to the mucous membrane. This worm measures from twenty-five to thirty feet when matured, and the segments of this tapeworm are wide and short, entirely different from the shape of the segments of the other tapeworms. The method of infestation is practically the same in all forms of tapeworms. The adult segments are extruded from the bowel of the host, are then swallowed by the hog, fish, or cattle, as the case may be. The ova hatch in the intestines of these animals, the larvæ penetrate into the muscles, live there, and become quiescent. The flesh of these animals is swallowed in an uncooked state by man; the embryo develops in the intestine; and the infestation of man is complete.

Sometimes there are vague nervous symptoms arising from the tapeworm, or there may be actual convulsions. The ordinary symptoms attributed to tapeworms such as increased appetite, jerky muscle symptoms and so on, are perhaps

rather imaginary on the part of the patient; indeed, they are simple signs of indigestion, without any particular reference to the worm which may be present. Occasionally links of the tapeworm are found in the vermiform appendix; the writer has notes of one patient who was operated on for repeated attacks resembling appendicitis and at the operation four links of tapeworm were found in the vermiform appendix. This particular case I am quite certain could not have been diagnosed anything other than appendicitis, yet the appendix itself at the operation was found to be relatively normal.

The blood of the host in infestation by the fish tapeworm, or *Dibothriocephalus latus,* shows a very extreme anemia, and resembles the blood-picture of pernicious anemia from which it can be differentiated only by finding the worm in the feces. There is usually, as is common with intestinal parasites of any kind, more or less eosinophilia in these cases.

The diagnosis is perfectly easy, and consists of finding either the ova or the links of the tapeworm in the feces. It is a great mistake to treat a person for tapeworm, or indeed for any other kind of intestinal worms, without being sure of the diagnosis. The examination of the feces should be made in every suspected case, and unless the ova or the links or the adult worm is found in the feces, no treatment should be given. As said in the beginning, the symptoms are extremely irregular from any kind of an intestinal parasite, and treatment cannot be safely based upon them.

Prophylaxis of tapeworm disease is most important. The first step should be the careful destruction of the feces containing the ova and the tapeworm. This can easily be done either by burning the feces or by mixing them with some solution such as chlorinated lime or strong solution of bichloride of mercury. The most important means of prevention is careful examination of the butcher's meat in the abattoir. In this meat fresh ova can be found if carefully looked for, and most Governments now have a regulation in which all meat is inspected, both beef and pork. According to Stiles, in inspection of meat special stress is laid upon the examination of the tongue and of the diaphragm, which are favorite seats for deposit of larvæ in the animals.

TREATMENT.

Perhaps as important a part of the treatment of tapeworm disease is preparation of the patient before any drug is given; indeed, the remedy is useless without this preparation.

At the beginning of the treatment, the patient should be given a brisk purge, preferably sulphate of magnesia or sulphate of soda. Following this purge the patient should be absolutely starved for 24 hours, nothing except water being taken into the stomach. At the end of 24 hours he should be given another purge, and, after this has acted, the selected vermifuge is to be given. Many drugs have been used for the cure of tapeworm. The one which has been most useful to the writer is aspidium, the oleoresin of male fern. This should be given in doses of 1 dram (4 mils), to be repeated in 2 hours. It is perhaps best administered in capsules, the wisest way to prescribe it being to give a prescription for the oleoresin and at the same time a prescription for empty capsules. The patient can fill these capsules himself, and take them. If they are bought in capsules in the drug store, the capsules are very apt to be soiled with the nauseating taste of the drug. Capsules containing the oleoresin of the male fern are manufactured, but are no more effective, and are more expensive than when prepared by the patient. Another method of administration is the fluid extract, a dram being mixed in sugar and then swallowed. This is not a very nauseating way of taking the drug. It is always best for the patient to remain quiet after taking the remedy, in order to avoid the nausea attendant upon its use. Two hours after the last dose of male fern has been administered, the patient takes another brisk purge of magnesia sulphate or sulphate of soda. This last stool should be received in a receptacle containing water, for the reason that a careful search must be made for the head, and the head is so small that it may be very readily overlooked in an ordinary stool.

Pelletierin, the active principle of pomegranate seed, is a very efficient remedy used for the tapeworm. This can be given in capsules, in doses of 5 to 10 grains (0.3 to 0.6 Gm.), repeated every two hours until three doses are taken. This also should be preceded by fasting and followed by a purge,

just exactly as is the male fern. Pumpkin seed is another remedy which has been used, and is particularly useful where the individual is old and weak. Two ounces (62.2 Gms.) of this may be made into an electuary by grinding up with sugar. It is not an unpleasant drug, and is quite effective. Perhaps the great reason of failure in treating tapeworm is failure to starve the patient a sufficiently long time, and to get his bowel entirely empty before the remedy is given. Unless the head is brought away the patient is not cured, and therefore careful search should be made. However, if the head is not brought away, it is no positive evidence that it has not been passed, and therefore active treatment should be discontinued, to be renewed on appearance of the links in the bowels.

SOMATIC TÆNIASIS.

This disease is due to the infestation of the human being with the larvæ of one of the tapeworms. The two most common tapeworms which give rise to cysticeria are the *Tænia solium* and the *Tænia echinococcus.*

Cysticercus cellulose is the name given to the infestation of the human being by the larvæ of the *tænia solium.*

The larvæ of *Tænia saginata,* or beef tapeworm, may infest man, but it does so very much more rarely than does that of *Tænia solium.* The symptoms of this disease depends entirely upon the organs infested by the larvæ. A general infestation has been described, in which the symptoms are miscalled those of "rheumatism"; the individual is sore, is stiff, aches all over and has fever, but on careful examination it is discovered that this so-called "rheumatism" is not an actual arthritis, but is due to soreness in the muscles; still more careful examination will show that there are small bodies under the skin, and if these are taken out, they will be found to be the larvæ of the tapeworm.

Nervous symptoms are due to the implantation of the larvæ in the spinal axis. Here the symptoms depend altogether upon the part of the brain or cord in which the larvæ are situated. There are cases on record in which large numbers have been found in the ventricles of the brain without giving rise to any symptoms whatever. Osler reports a case he saw

in which there were symptoms of diabetes and vague nervous symptoms. Cases with paralysis have been reported, where the cysticercus has been located in one of the active centers, the leg center, etc. The larvæ have been found and demonstrated by the ophthalmoscope during the life of the patient in the vitreous humor, giving rise to blindness and other derangements of sight.

ECHINOCOCCUS DISEASE.

This disease is due to the infestation of the human being by the larvæ of the *Tænia echinococcus*. This tapeworm is not a habitat of the intestine of man, but is found in the intestine of the dog. These are tiny tapeworms, not more than three or four millimeters in length. The hog and the ox are the intermediary host of this small tapeworm. The segments and ova of the worm are taken into the intestines of these animals, develop there, and find their way into the solid organs where they lodge. There is irritation and inflammation in the region in which these larvæ come to rest, and this finally gives rise to a cyst; after the cyst has arrived at a certain size, other "daughter cysts" develop on their walls; when these daughter cysts develop sufficiently, they also develop cysts on their walls. These are the granddaughter cysts.

Such cysts are filled with a colorless material. On the walls little granulations occur which are really the heads of the undeveloped tapeworms. These heads or *scolices*, present four sucking-disks and a row of hooklets. The scolices, taken into the intestine of the dog, are capable of developing an adult tapeworm. When these larvæ gain access to man they may lodge in any portion of the human economy. The *echinococcus* of the liver, the *echinococcus* of the kidney, the *echinococcus* of the nervous system are well known forms, and give rise to tumors in these positions, which develop pressure symptoms depending for degree upon the organ interfered with.

TREATMENT.

The treatment of echinococcus disease in man is, unfortunately, unsuccessful, except from the surgical standpoint. When a large cyst of the liver exists, it may be excised. If the

kidney is found to be the seat of an echinococcus cyst, either the cyst or the whole kidney may be removed, and the patient be perfectly well afterward. Upon the position of the cyst in the nervous system will depend the availability of surgical means of relief.

DISEASES CAUSED BY NEMATODES. (ROUND WORMS).

The most important round worms which infest man are:

1. *Ascaris lumbricoides,* giving rise to "Ascariasis."
2. *Oxularis vermicularis,* or threadworms.
3. *Trichina,* giving rise to "Trichiniasis."
4. *Ankylostomia,* causing hookworm disease (elsewhere described).
5. *Filiariæ* (elsewhere described).

ASCARIASIS.

Ascariasis is due to infestation by the *Ascaris lumbricoides,* a very common parasite, resembling very much the ordinary earth-worm, except that it is much lighter in color. It is much more common in children, but may be found in any age. Not only is this the most common worm that infests human beings, but it also yields more readily to treatment. Usually these worms are not numerous, but consist of only one or two, at each infestation.

Symptoms may be absent. Sometimes the first evidence of their presence is their appearance in the stool or in the vomitus. The symptoms upon which the laity lay much stress are picking of the nose and stomachache. These symptoms, however, if they are due to worms at all, are simply due to the indigestion which they cause, and may be present in any kind of indigestion. Without the knowledge of the worm being seen either in the stool or the vomitus, or the knowledge gained by the presence of the ova in the stool, children (who are the ones most frequently infested) should never be given vermifuge medicine. These worms may give rise to nervous manifestations, such as convulsions. They have been known to cause jaundice by plugging the common duct.

TREATMENT.

In beginning treatment for round-worms it is, perhaps, wise to put the children on a light diet and give them a purge of castor oil or citrate of magnesia. This, however, is not an absolute necessity. The drug which is absolutely a specific against round-worms is santonin. This drug may be given in 1-grain (0.065 Gm.) doses three times a day for three successive days, and then followed by a dose of calomel. I think this is much better than giving the santonin combined with calomel, because if the calomel is given with the vermifuge, the bowels are moved rather frequently, and the drug does not get an opportunity to come in contact with the worm. The stools should be carefully watched after this treatment, and if neither ova or worm appear in the stool, it should be repeated. The only untoward result of this dose of santonin is occasionally a yellow vision, which is simply annoying and has no serious import. Further infestion can be prevented by carefully washing the hands and manicuring the nails when they have come in contact with the anus.

OXYURIS VERMICULARIS.

These tiny worms, which appear at the anus, give rise to itching, and in females they creep into the vulva and vagina and cause a vaginitis. Occasionally these worms have been found in the vermiform appendix, that organ under such conditions being quite a large tumor packed with these tiny worms.

General symptoms are very uncommon, the chief one being pruritus ani. There are cases on record, however, where the worm has penetrated the wall of the gut and given rise to pericecal abscess. The worms develop in the small intestine, and then gravitate to the cecum, from which place, after the female worm has become gravid, they appear at the anus. The longevity of these worms is important in the treatment, because if one administers remedies simply to remove the worms from the lower bowel, there will very soon be a reappearance of the worm at the anus, because the source, the actual origin, has not been touched by these local applications to the rectum. The best method of treatment, it seems to the writer, is

first, to remove the worms from the anus and rectum, and then immediately direct one's treatment to the adult worms in the small or large intestine.

Local applications to the rectum of chlorid of sodium, (ordinary table salt), dissolved in water, or the injection of a decoction of quassia are quite efficacious in removing these adult worms from the rectum. The decoction of quassia may be made by directing the patient to purchase at the drug shop 1 ounce or 2 ounces (31.1 or 62.2 Gms.) of quassia chips, putting them in a pint of water and boiling to a half-pint (236 mils), and then using 2 or 3 ounces (60 or 90 mils) of this as an injection. This in a day or two will remove all the worms from the rectum.

Reinfection plays a very large part in the persistence of these small parasites, and therefore every individual who is infested with *Oxyuris vermicularis* should be provided with a tight muslin drawer without an opening at the genital parts. This prevents the child from scratching his anus when he is asleep or when he is awake and conveying the ova of the worm by his soiled fingers to his mouth where they are swallowed. Without this precaution the treatment of pin-worms is almost an interminable affair. For the destruction of the worms, in the intestine itself, the administration of santonin, exactly after the manner it is administered in round-worms, is beneficial. Thymol has been used and is efficacious, but in the opinion of the writer is not the remedy of choice.

TRICHINIASIS.

The adult worm, or *Trichinæ,* lives in the intestine of man, and gives rise to gastro-intestinal symptoms—diarrhea and intestinal discomfort. But this intestinal implantation is a small part, although a constant one, of the danger of trichinæ. The intermediary host is the hog. Hogs harbor the larvæ of the trichinæ in their flesh; the flesh in this condition, uncooked, is entirely unfit for man; thus the larvæ are swallowed, and in a very short time the adult trichinæ develop in the human intestines. This is the first stage of trichiniasis. The females, containing many thousand of living embryo, pierce the intestinal canal and probably deposit their embryo directly into

the blood and lymph-channels en route. From the blood-vessels and lymph-channels, the embryo invade the various portions of the body, particularly in the muscles, where they give rise to certain symptoms to be later described; they become encysted in a spiral form, and finally in the last stage, when they are entirely quiescent, become calcified.

The whole cycle of *Trichinæ,* as given by Stiles is as follows: First, man obtains trichiniasis from eating pork; second, hogs become infested from uncooked swill containing scraps of pork and from rats; rats obtain their infestion from eating each other, and eating scraps of pork in houses or meat shops. Rats alone, swine alone, or rats and swine together may keep up an endless chain of infestation. The infestation which reaches man may terminate through the death of the individual. Accordingly, man must be viewed as a more or less accidental host, while the rat, because of its cannibalistic habits, presents theoretically ideal conditions as the normal host.

After the uncooked pork containing the live larvæ is eaten, there is a period of quiescence lasting from several hours to a few days, and this is followed in severe cases by digestive symptoms—vomiting, pain in the abdomen, sometimes diarrhea, although these initial symptoms may be entirely absent. Within a day or two after the onset of gastro-intestinal symptoms, the patient develops fever, which may range as high as 104° or 105° F. (40° or 40.5° C.). This temperature range resembles closely the temperature of typhoid fever, and in sporadic cases, is very apt to be mistaken for that disease. The muscles become sore, swollen and tender, the patient becomes restless and very much disturbed. If the lungs are affected, there may be urgent dyspnea. If the infestation is very severe, the patient may die of exhaustion and fever.

The blood of an individual infested with trichinæ is very characteristic; there is a high leucocytosis, Dr. A. H. Mellersh and the writer having described 1 case in which a count of 32,000 leucocytes and an eosinophilia of 23 per cent. were present. The combination of high fever leucocytosis and eosinophilia, is quite characteristic of trichiniasis, although, as Da Costa has shown, eosinophilia is not present in cases of decided chronicity. When these symptoms present them-

selves, a small bit of the muscle should be excised, and search made for the trichinæ. The presence of the trichinæ in the muscle, will of course establish the diagnosis. If these cases occur in groups, after a repast or a picnic of some sort, where uncooked pork has been used, trichiniasis is naturally suspected, and the diagnosis very easily made.

Prevention in this disease is the most important part of the treatment. Care must be taken that no pork is sold that contains trichinæ. This, of course, should be done under government supervision. As Osler points out, this would be a very expensive and tedious operation, and perhaps would not be practicable in this country. On the other hand, if everyone would be careful and eat no pork except it be thoroughly cooked, this in itself would make trichiniasis an unknown disease.

TREATMENT.

When the case is seen in the very early stages, and intestinal symptoms are present, a brisk purge should be given, in order to drive as many of the adult worms as possible from the intestine. Even if one female adult is removed from the intestinal tract, this probably will prevent hundreds of larvæ from entering the circulation. Santonin and thymol must be given, to kill the adult worms. After the larvæ have entered the muscles, the treatment must be entirely symptomatic; warm applications, tepid bathing when the temperature is high, and supportive measures are methods worthy of use. The author has thought that probably the administration of neosalvarsan, when the larvæ have entered the blood-vessels might be of some value.

PARASITIC INSECTS.

PARASITIC ARACHNIDIA.

This troublesome insect in this affection, known as the itch mite, gives rise to very many annoying symptoms, although perhaps never fatal. Itching is the chief symptom. Infection by this insect is extremely common in India.

Usually there is herpes due to the first implantation of the insect. From these herpetic areas the mite burrows under

the skin, making tracks, which contain at the end the itch mite. This is the male, the female being rarely found. The individual infested scratches himself, giving rise to scratch marks which become infected, sometimes causing large ulcerating areas.

TREATMENT.

The important point in the treatment of these cases is the diagnosis. Very frequently real itch, particularly as it occurs in unclean persons, is taken for all sorts of skin diseases.

The treatment is exceedingly simple. A hot bath should be given and repeated every day, and the affected parts rubbed with precipitated sulphur, 20 grains (1.3 Gm.) of sulphur to an ounce (31.1 Gm.) of a simple ointment. Not only should the parts of the body where the eruption is seen be rubbed with this ointment, but all the portions of the body where the skin is thin, should also be anointed, particularly the skin of the thighs and under the arms.

IXODIASIS (TICK DISEASE).

Quoting from Osler the entire paragraph in his "Practice of Medicine": "In South Africa, particularly in the western provinces of the Uganda Protectorate, western districts of German East Africa, and the eastern regions of the Congo Free States, there is a disease known by this name believed to be transmitted by a tick, *Ornithodorous,* or *Argas Monbata.* Christy states that the bites of the *O. savignyi* do not produce any ill effects. The ticks live in old houses, and their habits are very much like those of the common bed bug. This tick transmits the *Spirochæta duttoni,* the cause of the African form of relapsing fever."

The Spotted Fever of Montana and California, Rocky Mountain Spotted Fever, is caused by a tick, *Dermacentor occidentalis.*

Pediculosis; Phthiriasis. These pediculi are of great importance, and particularly since it has been discovered that typhus fever is transmitted solely by the body-louse.

Pediculosis is due to the *Pediculus capitis,* insects which inhabit the heads of uncleanly individuals. Sometimes they are in immense numbers. One patient presented himself in the Out-

Patient Department of the University of Pennsylvania, in whom the hair of the head was actually teeming with thousands of head-lice, and yet this patient apparently had no knowledge as to what was the matter. The ova of these insects are contained in little sacs, called "nits," which are attached to the root of the hair by gelatinous material. They can be differentiated from the bits of dandruff and other foreign material by the fact that they are tightly adherent to the hair, and if put under a low-power microscope they are seen to be animal material, and when crushed, eggs will be seen in their contents.

Very frequently, when there are not very many of the lice present, they are more numerous near the nape of the neck, and an ulceration in this area, due to scratching, is a very suspicious sign that the head is infested with lice. If the hair be carefully examined in this condition, the lice or nits will be discovered.

The treatment of the head-louse is very simple. The hair should be shaved when the insects are very numerous, the whole scalp washed with coal oil, and enveloped in a gauze bandage for some hours. This will effectually kill all of the nits. Even after this treatment has been administered, scrupulous care must be taken that reinfestation does not occur. There is perhaps nothing more disturbing to a well regulated household than to see a child come home from school with a number of head lice. In these small infestations, it is not necessary to actually cut the hair off, but by careful combing and destroying all the nits, and then using a solution of carbolic acid, 1 to 50, or bichlorid of mercury, 1 to 500, on the hair, the insects and nits will likewise be destroyed.

Pediculus Corporis. This louse is larger than the head-louse; it is of particular importance because of its relation to typhus fever. It has now been shown that without the louse there would be no typhus fever. These body-lice attach themselves to the clothing, and therefore the best way to destroy them is to destroy the clothing by burning or by disinfecting it under hot steam. Often the body is covered with scratch-marks, sometimes with ulcerations, the result of infection of said scratch-marks. The itching, which is the result of the bite of these insects, can be controlled by warm baths, and by inunction of the body with carbolic acid ointment or by the ap-

plication of carbolic solution, 1 to 20, or bichlorid of mercury, 1 to 1000.

The *Pediculus pubis* is an insect somewhat smaller than either of the other lice, and inhabits those portions of the body where the hair is short, principally the pubis and under the arm-pits. Pubic lice are usually an indication that the individual has had some intercourse with another individual who is uncleanly in his habits.

The treatment is shaving the pubic hair and pubis, shaving out the axillary hair and anointing the axillæ and pubes with mercurial ointment. One must not be satisfied with shaving the hair and applying the mercurial ointment, but must wash the parts thoroughly in warm water.

CIMEX LECTULARIUS.
(Common Bedbug).

This insect lives in bedsteads which are not kept clean and in cracks of boarding about old houses which have been allowed to become unsanitary.

To some individuals they cause a great deal of annoyance, and others are not affected by a bite. Any bedstead or room which is infested with these insects, can be thoroughly cleansed by fumigating the room with sulphur, and taking the bedsteads apart and cleansing them with coal oil.

PULEX IRRITANS.
(Common Flea).

These are very common in certain districts, particularly in Italy, where practically everyone is bitten by fleas.

They give rise to no serious symptoms, but cause some irritation, and sometimes urticaria.

PULEX PENETRANS.
(Sand Flea, Jigger).

This little insect is found in the West Indies, particularly, where it bites and burrows under the skin. It gives rise to no serious symptoms and may be removed very easily by a needle.

PARASITIC FLIES.
(Myiasis, Myiosis).

The larvæ of various flies have been found in different portions of the body. A few worms or larvæ of the Lucilia macellaria, are often found in the nose and in wounds, and sometimes in the vagina.

Treatment consists in the removal of the larvæ by forceps. Larvæ of many different flies have been found in intestinal contents and in vomitus. They very rarely give rise to any serious symptoms, although almost all the individuals so afflicted complain of having "something alive" in their stomach, or in their intestine.

RABIES.

Hydrophobia is transmitted to man by the bite of various lower animals, particularly dogs, wolves and cats.

The disease is transmitted by the saliva with which the wound is contaminated when the individual is bitten. The bites are more frequently followed by the disease when they are on the bare surfaces, such as on the hands and on the face. When the bites are through the clothing, relatively few individuals develop the disease.

The period of incubation varies greatly, averaging perhaps from one to two months. After a person has been bitten by a rabid dog, the first symptoms are signs of irritation around the wound. The patient becomes nervous, irritable, and often introspective. Then there is irritation about the larynx, the patient often having difficulty in swallowing liquids. Soon any attempt to swallow liquid is followed by the characteristic respiratory spasm. If the patient is thirsty and attempts to drink, he tosses the liquid into the back of his throat, and at once goes into an apnoeic condition due to failure of the respiration, caused by a spasm of the respiratory muscles. Not only does the attempt to swallow liquids bring on these spasms, but a sudden noise, or being disturbed by a visitor, or a draft blowing on the patient may precipitate an attack. The temperature rises; the patient often becomes cyanotic, thrashing about the bed, and convulsions ensue. Toward the end

the individual becomes paralyzed, lies quiet, and usually dies in coma.

TREATMENT.

Prophylaxis is the most important part of the treatment. The methods to be followed are as follows: Immediately upon the patient being bitten by a rabid dog, the wound should be thoroughly cauterized by, first, an incision opening the wound widely, and then the use of a hot iron or nitric acid directly into the opened wound, the patient being anesthetized. This same cauterization should be done on every wound made by a vicious animal, supposed to be rabid. The animal should be then confined, and on the first appearance of rabies in the animal the stricken individual should be given preventative inoculations or Pasteur treatment. Many State boards of health now examine gratis the nerve-centers of animals suspected of rabies. The dog is killed, the head severed from the body, packed in ice or other refrigerating material, and shipped to the authorities. In the State laboratories the ganglia are examined. A certain diagnosis for or against rabies can thus be made, and treatment begun sooner than it can be if symptoms of rabies in the dog are waited for. This virus may now be purchased from almost any of the biological chemists, and from the New York Board of Health. The virus is sent to the physician in syringes or other containers, which may be used by anyone, even though he is not accustomed to the treatment. Pasteur treatment is based upon the fact that the virus of rabies concentrates itself in the nervous centers of animals sick with the disease. Rabbits are inoculated with the virus; when they develop rabies they are killed; the spinal cord is removed aseptically, and dried over caustic potash. Then this infected cord is emulsified, proved, and sent to the physician in the containers. The older cords are sent first, then daily doses are sent rapidly approaching the cord of one day. The contents of these containers is injected hypodermatically. Very few patients develop rabies after these prophylactic measures.

The cure of hydrophobia after the paroxysms have begun is entirely hopeless. The treatment should be directed to the control of the paroxysms. This may be done by the administration of chloroform, or keeping the patient under the influ-

ence of morphin and chloral and bromid of potassium, and making his miserable existence as comfortable as possible. He should be in a darkened room, and with very few visitors. The nurse should be carefully clothed in a gown, have her hands encased in gloves, being careful not to inoculate any abrasion with the saliva of the patient.

PSITTACOSIS.

This is an infectious disease transmitted to man from birds, and particularly from parrots. According to those familiar with the disease, it resembles typhoid fever complicated with pneumonia. The diagnosis from typhoid fever can be made, however, by the absence of the Widal reaction and a negative blood culture for typhoid bacilli. It is certain that this severe disease, with rather a high mortality, has occurred in epidemic form in houses whose occupants have been owners of various feathered pets, notably parrots.

The treatment according to Boggs is the following:

TREATMENT.

"In view of the possible agency of the parrot in conveying this infection, the handling of the sick birds should be discouraged. Cleanliness and disinfection of the cages should be carefully observed. The patients should be isolated as a precautionary measure. Tub baths and cold packs are highly recommended by the French observers, who treat the cases throughout like typhoid fever."

MILK SICKNESS.

Milk sickness, or "trembles," as it is called, is a disease transmitted to man from cattle, sick with this disease.

The disease, as said, is primary in cattle, and is due to the *Bacillus lactimorbi.*

The symptoms in man are weakness and loss of appetite, soon followed by gastro-intestinal symptoms—vomiting, diarrhea, and abdominal pain. The breath has a sweetish odor, the tongue becomes swollen, the pulse is quick and full, and

there are marked nervous symptoms, the result of the toxemia, convulsions and delirium being very common. The cases are remarkably fatal in man.

TREATMENT.

The important point in the treatment is prevention. As this disease is always transmitted from cattle, care should be taken that the milk of the cows, or the flesh of any of the animals affected by trembles is not used.

The treatment is entirely symptomatic. The patient should be in bed, and, if the fever is high, hydrotherapy should be used. Magnesium sulphate and castor oil should be used as purgatives to clean out the intestine of the infected milk.

Bromid of potassium, for the nervous symptoms, is useful. An abundance of water, by the mouth, by the rectum in the form of Murphy drip, and, if necessary, by intravenous injection of normal salt solutions.

FOOT-AND-MOUTH DISEASE.

This disease, occurring in cattle, sheep and hogs, is transmitted from these animals to men direct, apparently by transmission.

The affected cattle rapidly lose flesh, and become feverish, vesicles occur along the edges of the tongue, and desquamation of the entire tongue, often in large patches, sometimes of the entire tongue, is common. In cows there are vesicles on the udders and on the teats, and there is profuse salivation. The mortality is not high in cattle, but often whole herds are ordered slaughtered in order to prevent an epidemic of this disease from spreading.

During the course of the disease men sometimes become infected, and show exactly the same symptoms as the cattle; they have ulcers in the throat and on the tongue, with fever and emaciation. Usually the cases are not fatal, but sometimes there is considerable toxemia, and in one epidemic quoted by Osler there were eighteen deaths. When an epidemic prevails in cattle, the milk should be boiled. It is not thought that the meat of infected cattle transmits the disease.

TREATMENT.

The methods of treatment recommended in man by Boggs is to isolate the patient, and to apply local remedies to the ulcers. Permanganate of potassium is recommended as a mouth-wash. The ulcers may be touched with silver nitrate, or with copper sulphate. Drying powders may be applied to the external lesions. Special attention is given to the diet, and in severe cases and in young children feeding by a nasal tube, a stomach-tube, or rectal enemata may be indicated.

INFECTIOUS COLDS.

Unquestionably so-called colds are an infection due to one or the other of several micro-organisms.

The *Micrococcus catarrhalis,* which affects the mucous membrane of the nose particularly, has been cited as the causative factor of many of these attacks, but cases which are indistinguishable, except by cultural methods, are found due to the pneumococcus, the influenza bacillus, and to other micro-organisms.

The symptoms of these colds are chilly sensations, coryza, lachrymation, short hacking cough, and a laryngitis, together with aching of the limbs and headache. Here, again, there is danger of considering these infectious colds nothing but colds, and hence treating them lightly, when as a matter of fact they may be the premonitory symptoms of measles, or of bronchitis, which may run on to a serious proportion, and, indeed, to almost any infection of the respiratory tract.

It is difficult to have our patients obey the order "to bed" in these light attacks, but certainly that is a measure of safety, and it should be advised to all our patients thus affected.

TREATMENT.

Rest in bed, with the rooms well ventilated, or even in the open air, the patient being well protected, is the most important of all of the points of treatment. The coryza may be controlled to a certain extent by the use of some alkaline spray, such as Dobell's solution, or a normal salt solution. Adrenalin chlorid solution, 1 to 2000, may be sprayed in the

nostrils, and often will give quite instant relief. The same application may be made to the throat. The aching is best controlled by one of the salicylates, acetylsalicylic acid or aspirin being the choice of the writer. Phenol salicylate or salol is another excellent drug; or a combination of phenol salicylate and phenacetin is comforting, and often in a few hours will convert a very uncomfortable individual to one who feels badly, but is not very acutely ill. This combination should be given in 5-grain (0.32 Gm.) doses of each drug, repeated every two hours, and continued for at least twenty-four hours. The headache and fever are also controlled by this medication.

If the cough is dry and hacking, small doses of an opiate, such as paregoric, will often control it so that the patient gets comfort and rest, instead of being uncomfortable and coughing.

The bowels should be opened by small doses of calomel, $\frac{1}{10}$ of a grain (0.006 Gm.) every half-hour until a grain (0.065 Gm.) is taken, or a saline, such as citrate of magnesia. Cocain is mentioned in order that it may be avoided. It is a very dangerous habit to give these patients a spray of cocain for the nose, for many cocain habitués have been made in this way.

MILIARY FEVER.

This disease has been known for centuries, and in the early fifteenth and sixteenth centuries was quite a fatal disease, particularly in England. At present it seems to be limited to very slight local outbreaks, particularly in Switzerland, and lately epidemics in Austria have been reported.

It is a disease characterized by sudden onset, high fever, and a remarkable amount of sweating, the sweat continuing with the fever, and throughout the entire course of the illness.

The temperature ranges from 102° to 103° F. (38.8° to 39.4° C.). The heart is rapid, palpitation being one of the symptoms which annoy the patient. Nervous symptoms—delirium and coma—are very common. The eruption is best described by Boggs:

"The eruption appears on the third or fourth day, and is seen first on the neck and back, under the breasts and axillæ,

and between the thighs. A general erythema is present, in addition to which appears (*a*) sudamina (miliaria alba or crystallina), (*b*) red papules becoming vesicular (miliaria rubra), (*c*) petechiæ of variable size (purpura miliaria). The appearance of the eruption is preceded by itching. The red papulovesicular type is most frequently observed, with usually some sudamina; the purpuric type is not seen so often, and occurs in the cases with other hemorrhagic manifestations. Vesicles are also frequently found on the mucous membranes of the conjunctiva, nose and mouth. The rash may come out rapidly, and cover the whole body in twenty-four hours, or slowly, and in successive crops. The desquamation may be advanced in the regions first affected, while other areas show the early stages."

The treatment is entirely symptomatic. The patient should be in bed. Hydrotherapy should be practised, and the patient should, of course, be isolated. On account of the extreme sweating throughout the entire course of the disease, the clothing of the patients has to be repeatedly renewed to keep them comfortable. Notwithstanding the profuse sweat, hydrotherapy is of the utmost importance, relieving many of the symptoms of the disease. The nervous symptoms can be best controlled by the bromids in large doses, 20 grains (1.3 Gms.) every two or three hours for forty-eight hours at a stretch. When the delirium is violent, the patient must be controlled by morphin. Atropin has been used to control the sweat, without, however, very much effect.

The convalescence is often prolonged. The patient should take an abundance of food, rest and fresh air during the entire convalescence.

Exanthemata

BY

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Exanthemata.

FOREWORD.

In the management of contagious diseases the physician is confronted by a twofold obligation—his duty to the patient and his duty to the community.

The physician's duty to the patient, of course, consists of doing everything which tends to recovery, and to the avoidance of harmful after-effects of the disease in question.

It is unfortunately true that, with the exception of diphtheria, we have at our command, in no instance, a remedy for a contagious disease which is in any sense of the word specific. On the other hand, in the most widespread and loathsome of all the contagious exanthemata—smallpox—we have a certain prophylactic against its spread. Vaccination against smallpox has conferred immunity upon millions of people, an immunity of such definiteness and length of duration that were vaccination universally practised smallpox would be unknown.

The physician's duty to the community means the bending of every effort to avoid the spread of disease from his patient to other persons. This is not a matter of such difficulty as might appear if all possible precautions be taken.

With the exception of smallpox and chicken-pox, it is very certain that the contagious diseases are not often air-borne; even in these instances the question is a matter of some doubt, but we are not without evidence that there may be such a possibility.

It is known that the infection of all of the exanthematous diseases may be spread by an intermediate carrier. Probably this is more often true of scarlet fever, diphtheria and smallpox than of the other contagious diseases. But in the vast majority of instances all of the contagious diseases are spread by the direct communication between the patient and some other individual not as yet infected.

In order that the physician may guard against the spread of contagion not only are the foregoing facts of the utmost importance, but some knowledge of the location of the infecting agent in the diseased person himself is of absolute importance. In other words, to prevent the dissemination of infectious material we must first form a definite idea of what this material is.

Briefly, our knowledge upon this important subject may be summarized as follows, stating only proven facts and with no regard to theoretical considerations of infection:

In diphtheria we know that the infecting agent can reside in the discharges from the site of the lesion, either during the active process of the disease or during and after convalescence.

In scarlet fever the discharges from the nose, throat, ears and suppurating glands carry the active infecting agent, which is not found in the desquamating skin.

In measles the discharges from the nose, throat and eyes are infectious during the stage of invasion, and while the disease is at its height.

The infecting agent does not reside in the chronic aural or nasal discharges of a case of measles. It is known also that the lesions of measles harbor the virus in a form which permits its transmission. The virus of measles is extremely short-lived, being rarely active after the acute stages are passed.

In smallpox and chicken-pox the local lesions are the only demonstrable locations of the active infecting agent.

With these facts in mind, it is evident that effectual isolation of all sufferers from contagious diseases and the isolation of all contacts until the period of incubation is definitely over would, in all probability, cause a practical disappearance of these diseases.

But while this is true in theory, in practice it is a matter of the utmost difficulty to carry out preventive measures based upon the foregoing premises. For this there are a number of reasons.

The most important factor in preventing the quarantine of contacts, aside from the disinclination of the public to submit to drastic regulations, is the contagiousness of these

diseases before the specific lesions manifest themselves, *i.e.*, before a diagnosis can be made. This being the case, it would seem that a radical suppression of these diseases in their entirety can be hoped for only when some form of prophylaxis, such as vaccination for smallpox, is found.

When, however, the disease in question is so far developed that a correct diagnosis is possible, isolation of the patient is of the greatest importance in preventing further spread of the disease.

There is no doubt that treatment in a special hospital is the best method for protecting the community against the spread of contagious diseases, but this is not always possible. It is only in the larger communities that even partly ample facilities are at hand.

There are several factors in the handling of contagious diseases that furnish us our basis for the practical solution of the problem of effectual isolation.

The discharges from the patient carry the infecting agent. Therefore, our first problem must be to see that such spread is not possible. Bandages, dressings, gauze used for handkerchiefs, etc., are destroyed. Other articles of use or clothing are properly sterilized. Utensils are kept for the patient only, and proper precautions are taken as regards their freedom from infectious material, if they are taken from the sick-room itself.

The patient's attendants must exercise the utmost caution. The nurse should be isolated with the patient as far as possible. The attending physician should have his ordinary street clothing suitably covered when he enters the sick-room, and this covering should be removed upon leaving the room. The most important matter of all, probably, is the care of the hands. Anyone in attendance, on leaving the sick-room, should wash his or her hands each time, and the physician should supplement this by again washing his hands before approaching the next patient.

The importance of fresh air and ventilation to the patient is self-evident. On the other hand, the possibility of air conveyance of contagious diseases is sufficiently great to cause us to take certain precautions in this direction also. The sick-room should, by perference, not open directly into other

occupied rooms, and ventilation should be from outside of the house rather than from within.

It is doubtful in the extreme whether insects carry any of the contagious diseases, but to avoid all possible risks, it is proper that the windows of the sick-room should at all times be properly screened.

The value of sterilization of clothes and utensils after a patient's recovery is unquestionable, as is also the necessity of a cleansing bath before discharge.

Fumigation of rooms is of a more doubtful status, but this should be employed in addition to subsequent ventilation for a day or more.

In conclusion it may be remarked that the physician must be guided, not only by his own opinion upon all these matters, but should always follow absolutely the rules of his local health authorities in the management of all the details of quarantine and preventive steps.

SMALLPOX.

Smallpox is an acute contagious disease, characterized by a specific eruption and fever.

The etiology of smallpox is unknown. There is no doubt that the disease is caused by a micro-organism, but the specific cause has not as yet been isolated. The virus is contained within the lesions of the disease, and is very tenacious of life.

The mode of dissemination of smallpox is usually by direct contact between the patient and a non-immune person, but actual contact is not necessary to infection. There seems, however, to be ample evidence that the disease may be carried by healthy third persons and by fomites, even when a long time has elapsed after the exposure. The disease may be contracted by merely entering a room or building in which it exists, or even the immediate vicinity of the building.

Of all the contagious diseases, smallpox presents the best evidence of being at times air-borne and of occasional conveyance by insects.

Both sexes and all ages are prone to the disease, unless immunity has been acquired by a previous attack or by vac-

cination. The latter does not confer absolute immunity for more than a limited time, but even after this time it so modifies an attack that it becomes very mild.

The period of incubation of smallpox is from ten to fourteen days. In respect to the definiteness of its behavior after infection has occurred, it is unique among the contagious diseases.

The onset of smallpox is abrupt, the initial symptoms being nausea and vomiting, with chills and fever, accompanied by severe headache and backache, the latter two being the most constant symptoms. The prodromal fever is very high from the beginning, often reaching 104° F. (40° C.) within the first twenty-four hours after the onset. The actual smallpox eruption occurs on the third day after the onset, although prodromal rashes, erythematous or petechial, are occasionally noted a day or two previously. With the appearance of the rash the fever subsides, the toxic symptoms abate, and the patient feels quite well. The rash is at first macular, but in a few hours these macules become distinctly raised or papular. Within twenty-four hours after the appearance of the lesions vesiculation appears, and at the end of forty-eight hours it is complete. The vesicular stage lasts four days, after which the vesicles become pustules. With pustulation the secondary fever begins, and continues until the pustular stage is over. On the ninth or tenth day of the eruption the pustules are fully developed and begin to break, discharging a foul, purulent material. Thus scabs are formed. These usually begin to separate about the fourteenth day of the eruption. In the milder cases the entire process will be complete within three weeks. In severe or confluent cases it may be a matter of many weeks or months. Each lesion finally leaves more or less of a pit or pock-mark in the skin.

The complications of smallpox most commonly met with are laryngitis, due to the presence of the lesions on the mucous membranes, bronchitis, and occasionally bronchopneumonia. Frequent local complications are the formations of superficial abscesses and boils. Orchitis may occur, and this is a most severe complication. Eye complications, such as conjunctivitis, keratitis, and even panophthalmitis, may occur. Otitis media and adenitis are occasionally met with.

The diagnosis of smallpox is not difficult in a classic case. During the prodromal stage it must be differentiated from influenza and lumbago, and, when prodromal rashes appear, from scarlatina and measles.

In the stage of eruption the disease may be mistaken for measles, chicken-pox, syphilis, and certain drug rashes.

TREATMENT.

The treatment of smallpox is entirely symptomatic. In the early stages, the pain in the back and headache must be relieved, and for this relief an opiate may be required. During the whole course of the disease the patient should be treated as any other fever case. It is the practice of some who have had considerable experience in this disease to allow somewhat liberal a diet in the period between the primary and secondary fever. This is not necessary, but does not appear to be harmful. The liquid diet, principally milk, should be plentiful and continued while the fever lasts, and subsequently it may be gradually increased.

Elimination must be aided, the bowels kept open, and possible interferences with kidney function forestalled by the use of large quantities of water, and, perhaps, potassium citrate in doses of 15 to 30 grains (1 to 2 Gms.) every fourth hour.

There is no drug which either aborts or alters the attack. It should be noted that vaccination performed immediately after exposure either prevents or lessens the severity of the disease. If the evidences of toxemia be severe, stimulants may be needed, such as alcohol or strychnin.

Various measures have been advised to prevent pitting of the lesions, but none has been effectual. Neither light nor darkness, or any local application seems to have any effect. A carbolized vaselin or boric ointment dressing will allay irritation and help the loosening of scabs.

Attention to the eyes is essential. The use of a boric acid lotion and the cleansing of the lids are most important. Headache is best treated by the use of the ice-bag. The coal-tar products might, with advantage, be used in certain cases.

In cases of severe backache hot applications may bring relief. Insomnia and delirium are oftentimes a troublesome factor in the treatment, and are best controlled by bromids and chloral or veronal.

Every case of smallpox should be given as complete and prompt isolation as possible, and to accomplish this the patient should be sent at the earliest possible moment to an isolation hospital. Because of the fact that the disease can be conveyed through the air, the hospital should be situated as far as is practicable in a sparsely settled or uninhabited locality. It is a well-known fact that in certain instances smallpox hospitals have apparently formed foci of this disease. The most rigid quarantine must, then, be instituted and maintained.

All contacts should be vaccinated as quickly as possible, and thereafter kept under close observation until safely past the incubation period. Actual quarantine will be called for in instances where evidences of a successful and recent vaccination are lacking. The successful vaccination or re-vaccination of contacts, if done within the first three days after exposure, will usually prevent altogether an attack of smallpox; if the operation be done even as late as the sixth day after exposure it will modify an attack.

The movements of the patient for two weeks prior to the time of falling ill should be carefully investigated, with the idea of ascertaining if possible any connection with previous cases yet unreported. Similar efforts should be put forth with regard to all those with whom the patient has come in contact since falling ill, in order to discover any new cases and thus prevent new foci of infection.

The danger of a smallpox epidemic growing out of any particular outbreak will be in direct proportion to the percentage of unvaccinated individuals in that community.

All articles of negligible value that have been in contact with the patient should be destroyed; all articles worth saving should be disinfected. The thorough fumigation, cleaning, and, as far as possible, refurnishing of the patient's home or apartments should always be done.

The patient should be considered fit for discharge when the last scab has separated from the skin, and when all cores have been removed from the hands and feet.

VARIOLOID.

Varioloid is true smallpox in modified form, and is the name given to all very light or abortive cases. By some authorities the term is restricted to include only those cases in which the modification has been brought about by a more or less remote vaccination.

The course of varioloid is shorter and milder than that of the other forms of variola. The symptoms are often very difficult to recognize because of their mildness or aberrancy, and sometimes the condition is never suspected until secondary cases arise. The prodromal symptoms may be exceedingly severe and the rash very profuse. In every instance, however, the course of the disease is short, the eruption aborting in the vesicular stage and never going on to pustulation. There is no secondary rise of temperature, and the attack ends with the drying of the vesicles.

The *treatment* of varioloid is the same as for any of the other forms—purely symptomatic. Care should be taken, however, not to modify the quarantine. These cases are capable of giving rise in other individuals to any of the other forms of variola, even the confluent or hemorrhagic types.

VACCINATION.

Vaccination is the production of a localized vaccinia or cowpox. It is accompanied by certain febrile manifestations and other evidences of systemic infection.

A successful vaccination will prevent smallpox for a period of time not the same in all individuals. It varies from several years to a whole lifetime. For the first six years the immunity will be absolute in every instance. For longer periods of time it will cause a supervening smallpox to be more or less mild, the so-called varioloid (*q.v.*).

Technic of Vaccination. The part selected for the operation usually is the outer surface of the left arm, just below the insertion of the deltoid muscle; in females the leg below the knee is the site preferred. After thorough cleansing with soap and water, and the subsequent wiping off with alcohol the part is allowed to dry. Scarification is then done with

a blunt lancet, needle, or one of the many instruments especially devised for this purpose. Three or 4 parallel abrasions $\frac{3}{16}$ in. long should be made, penetrating the skin just deeply enough to show serum, but not to cause bleeding. The inoculation should then be made by dropping the virus on the wound and rubbing it in cautiously so as not to cause further flow of serum or blood. A temporary shield, easily made from a small piece of stiff paper and adhesive plaster, should be applied, with instructions for its removal the following morning. The shield is used for no other purpose than that of preventing infection or the rubbing off of the virus by the clothing. Its use otherwise might prove a detriment. The only local treatment that will be required after this, except in the event of secondary infection, will be the painting of the site twice daily with a solution of iodin 1 part, picric acid 4 parts, and alcohol 95 parts. This should be commenced two days after operation. Not only will it lessen the chances for outside infection, but it will also cut short the acute stage of the process.

If the vaccination is successful, or "takes," a small papule surrounded by a reddened area will show itself in from three to six days. In a few days more vesicles will form with a central depression and surrounded by a reddened and indurated area. The vesicles increase in size for several days, and then dry or break, leaving a quite adherent scab, which separates after a few weeks more. The scar at first is pink; later it becomes white, with numerous small pittings.

Constitutional symptoms are present in the majority of cases. They are malaise, anorexia, headache, and fever. Restlessness and gastro-intestinal disturbances are often noted in smaller children. Very severe symptoms should always bring to mind a possibility of secondary infection.

Vaccination should never be looked upon lightly by the physician. The strictest asepsis is called for at all times on the part of doctor, patient, virus, instruments, and dressings.

Vaccination is one of the greatest blessings given to humankind by the medical profession; nothing by the profession should be done to discredit it.

In the face of an exposure to smallpox, or even in the midst of an epidemic, there are no contraindications to vaccination. In the interest of public health, if for no other reason, the

physician should practise at every opportunity vaccination and re-vaccination. Were this possible with all peoples, smallpox would disappear from the face of the earth.

The Complications of Vaccination. A successful vaccination, properly carried out with due regard to asepsis, and with proper lymph, never offers complications. The severity of the local and systemic reactions varies, but neither requires treatment.

All the possible complications of vaccination are caused by secondary infections—either at the time of vaccination by contaminated lymph, by failure in asepsis, or by subsequent contamination of the wound.

At the present time, due to care in manufacture, there is no contaminated lymph.

A failure to observe strict asepsis at the time of vaccination may lead to the development of any of the secondary conditions, such as are commonly found in ordinary wound infections. The same holds true of infections subsequent to the vaccination, when scratching with dirty fingers is a common cause of complications.

Tetanus has resulted after vaccination, as has also syphilis, but these are rarities. Erysipelas and cellulitis are more common. Perhaps the most common and troublesome result of the ordinary infections of vaccination is the delayed healing of the site of the pustule, a discharging area often persisting for weeks or months.

The treatment of the complications of vaccination is exactly the same as when they occur after other abrasions or surface wounds.

VARICELLA.

Varicella, or chicken-pox, is a highly infectious disease of moderate intensity, and characterized by fever and by a specific eruption.

That varicella is a disease separate and distinct from variola is now universally admitted. Controversy regarding its identity with variola, however, raged throughout the medical world for several generations, as late even as 1870-73, during the great epidemic of those years.

It attacks individuals of any age, although it is encountered chiefly in children, especially in those under the age of ten. It is my experience that adults are more infrequently the subject of this disease than of any other of the acute exanthemata.

It is most highly infectious; in fact, next to variola, it is the most infectious of all these diseases. In studying outbreaks of this disease in the wards of diphtheria and scarlet fever hospitals, it is my experience that in almost every instance secondary cases will develop. I cannot say the same of other diseases, not even of measles. It is also the one disease which, in spite of the most painstaking precautions, will go from ward to ward, and from building to building even. This forces the conclusion that it is spread by ways other than direct contact. I feel safe in saying that the air, third parties, or inanimate objects may be means of disseminating this disease.

Varicella is endemic in cities and the larger towns, but it does not take on the form of large epidemics, as is the case with variola. The specific micro-organism is supposed to reside in the local lesion. The virus is rather short-lived, being in this respect markedly different from the virus of variola.

Varicella is a disease of the colder months, beginning, as a rule, in middle autumn, and lasting into the late spring or early summer.

The symptoms of varicella are, as a rule, fever and a rash. In private practice the rash in most all instances is the first symptom. In institutions, however, where closer observation is possible, it often happens that a moderate fever, 99° to 101° F. (37.2° to 38.3° C.) precedes the rash by a few hours.

The eruption, if seen within the first few hours after its appearance, consists of small papules which disappear on pressure. These develop almost at once into vesicles. The lesions vary greatly as to number; in some cases there may be so few as half a dozen; in other instances the lesions may number several hundred. The fever may go as high as 104° F. (40° C.).

Complications are very few. Secondary infections, through the local lesions, are the only ones that I have observed.

Severe conditions mentioned by some authors as complications I feel sure are mere coincidences.

Recovery is the unvarying rule in all uncomplicated cases. All fatalities that have come under my observation I have been able to ascribe to some cause other than varicella. The gangrenous type of the disease, which is the fatal form, is uncommon, and is seen in children already the subjects of some extremely debilitating condition when attacked, such as scarlet fever, diphtheria or tuberculosis.

In typical cases the diagnosis is extremely easy. In certain borderline cases, however, particularly in adults, it is impossible to differentiate with absolute certainty. The only condition of importance, with which such cases may be confused, is variola, especially the milder form, or those varieties modified by more or less remote vaccination. As is the case with German measles, it is absolutely imperative that the utmost care be taken in every instance to arrive at a positive and a safe diagnosis. Failure to do so may result in disaster to the community in a public health and business way, and also may impair irretrievably the usefulness of the family doctor in that particular community.

A scarlatinaform prodromal rash is encountered occasionally with varicella. A reasonable amount of effort should suffice to make a differentiation from scarlet fever possible in all cases.

In differentiating these diseases the following factors should be studied most carefully:

1. The movements of the patient for at least two weeks prior to the appearance of the rash, in an effort to establish connection between patient and some previous case.

2. The prodromal symptoms.

3. The character of the lesions, their distribution, and the history of their development.

4. History and evidences of vaccination, bearing in mind the influence a successful vaccination will exert in modifying variola, or in preventing it even, especially if the vaccination be so recently done as within the previous half dozen years.

TREATMENT.

Next to German measles there is none of the acute exanthemata that requires so little treatment, which is purely symptomatic. Rest in bed during the febrile stage, and until the lesions are well past the acute stage, should be insisted upon in all cases. A light diet and some mild fever mixture should be prescribed if the fever be high. To prevent secondary infection, which constitutes the only real menace of the disease, it is necessary that the hands be kept clean, and that they be securely tied in well padded mittens or stockings. It may be necessary to restrain even the hands, arms or legs. Itching should be treated with tepid baths, or by sponging with alcohol well diluted with water, or with vinegar-water, 1 part to 3 parts. After that any bland dusting powder should be employed. A good one is: Mentholis, 1 dram (4 Gm.); zinci oxidi, 1 dram (4 Gm.); pulv. amyli, 1 ounce (32 Gm.); talci, 1 ounce (32 Gm.). Secondary infections call for the same treatment as would any other similar surgical condition.

The patient should be considered ready for discharge when separation of the scabs is complete. In the average case, free from secondary infection, three weeks time will be required.

SCARLET FEVER.

Scarlet fever is an acute, contagious, self-limited disease, characterized by a diffuse scarlet eruption and a pharyngeal inflammation.

Susceptibility varies with age and with various other factors. Immunity is usually conferred by one attack. Children are far more susceptible than adults, but even all children apparently are not susceptible. A basis upon which such comparative or absolute immunity may be explained has not been found.

The etiology of scarlet fever is obscure. Its contagiousness, its acute febrile character, and its septic complications all point to the belief that it is of bacterial origin, but a specific organism has not as yet been demonstrated.

In most cases the mode of contagion is undoubtedly by direct contact. The disease may, however, be spread by

intermediate carriers, either persons or inanimate objects, such as clothing, carpets, books and toys. The possibility of the transmission of the infecting agent by insects is to be borne in mind when considering quarantine and prophylaxis. The disease is contagious in all of its stages; most highly so when it is most active. The infecting agent resides in the secretions, and of the greatest importance in this connection are the oral and nasal secretions and, in complicated cases, the discharges from the ears. The desquamating particles of skin, unless contaminated by secretions, are not contagious.

The period of incubation of scarlatina varies from one to eight days; it is usually from two to five days.

The onset of scarlet fever varies to a great extent with the severity of the attack. The invasion is abrupt, with rapidly rising temperature from 101° to 104° F. (38.3° to 40° C.). Children often vomit and show signs of general depression. Sore throat will be complained of in almost every instance, the pharynx and tonsils show congestion, and very early in severe cases a pseudomembrane appears. The tongue at first is covered with a white, furry coating, through which the swollen, red papillæ may be seen to project, and within a day or two the tongue begins to shed its coating, and by the fourth day will be clean, red and glistening, with the papillæ distinctly prominent,—the "strawberry tongue" of scarlet fever. The consistency with which the tongue thus desquamates in scarlet fever is, in certain cases, one of our most important diagnostic aids.

Usually within the first twenty-four hours after onset the rash appears, beginning on the neck and upper chest and spreading rapidly over the entire body. It is a distinctly punctiform erythema, and in typical cases varies in tint from a light pink to a light or dark red in color. It may be so slight as to be barely visible. In atypical cases it may assume a macular or papular appearance, simulating very much that of measles. Especially is this so about the wrists, ankles and the dorsa of the hands and feet. It is sometimes petechial, and miliaria as minute white vesicles are often seen. The rash may be limited in extent, being confined to the chest, abdomen, groins, axillæ, or the bends of the elbows. It may be entirely absent in very mild cases.

The diagnosis of scarlet fever depends upon the peculiarities of the rash and the pharyngeal lesions. Variations in the rash make a correct early diagnosis at times impossible. Indeed, in very rare instances an undoubted scarlet-fever infection, as evidenced by the incidence, course and pharyngeal symptoms, has been recognized in the absence of a rash at any time during the course of the disease. The throat condition may be such that a differentiation between scarlet fever and diphtheria will be impossible on the clinical evidence alone. In such cases it is only after repeated bacteriologic examinations that a positive diagnosis can be made. The possibility of a double infection of scarlet fever and diphtheria should be borne in mind. The greatest problem in the differential diagnosis of scarlet fever is the differentiation from rubella. The characteristic rash, fever, severe onset, tongue and pharyngeal symptoms of scarlet fever, and their absence in rubella generally make the diagnosis possible within one or two days. Perhaps the most difficult problem in the diagnosis of scarlet fever is the recognition of the very mild cases. Unless we encounter them in an institution or during an epidemic when every case of fever in a child is most closely examined, it is very easy to overlook these cases. Especially is this true where, with a mild febrile course, sore throat is not complained of. In a cursory examination a slight rash easily could be overlooked, and if it fades rapidly within a day or two a correct diagnosis would be impossible. Such cases are particularly important because if proper care is not taken of them a nephritis is likely to supervene; moreover, such cases, not being quarantined, will spread the disease.

The clinical course of the disease varies with its severity. We may classify cases of scarlet fever as: (1) *Mild cases,* those in which the course of the fever is from 100° to 103° F. (37.7° to 39.4° C.), the pharyngeal symptoms not very marked, and the signs of general infection and prostration slight. In these cases the rash fades very rapidly, beginning on the third or fourth day, and disappearing entirely by the fifth day. (2) *Moderately severe cases.* In these the onset is marked by a greater severity. The temperature goes up to 104° or 105° F. (40° or 40.5° C.), the rash is more intense,

and does not begin to fade until the fourth or the sixth day. The throat symptoms are more marked, and an exudation may be encountered. The systemic manifestations of the disease are marked, but recovery, apart from complications, is the rule. (3) *Severe cases,* those with a sudden onset and a rapidly appearing, widely spread and intense rash. Ulceration of the throat is common, and a cervical adenitis always accompanies it. Rhinorrhea is marked. The fever is high, either continuously so or of the septic type, and persists for several weeks. The symptoms of general sepsis are marked, delirium and restlessness the rule, and the pulse is rapid and often weak. Gangrenous changes in the throat may be encountered. In the fatal cases death usually takes place from sepsis alone, from complications, or from exhaustion. (4) *Malignant cases,* those in which death occurs from an overwhelming toxemia within a short time after the onset of the disease. These cases are seen only in the courses of severe epidemics.

The pathologic changes in scarlet fever may be described as consisting of a dermatitis (erythema), and an inflammation of the pharyngeal structures of varying degrees of intensity, with a lymphoid enlargement throughout the body in general.

TREATMENT.

The treatment of scarlet fever comprises: (*a*) such handling of the case that the spread of the infection is minimized; (*b*) the symptomatic and general treatment of the disease itself; and (*c*) the anticipation and the care of the complications. The treatment here outlined is, of course, that which is possible for a case treated at home. In institutional work the problems of isolation and management are somewhat more complex.

Isolation. Every scarlet-fever patient should be isolated. Wherever possible the patient should not only be kept alone in a room, but should have a whole floor, preferably the upper floor, or the one most distant from other persons, and, if possible, one with a door opening to the outside. Separate toilet facilities are desirable, and should be insisted upon whenever possible. The hanging of sheets soaked in carbolic acid or in bichlorid of mercury solution has been suggested by

many, but this precaution is not necessary. The only advantage of such a procedure is that it serves as an ocular reminder that a quarantine is on, and should be respected. All isolation will not be effective, however, if the attendants and nurses do not take every possible precaution. Those entering the room should put on a cap and gown and remove them at the door when leaving. Here we must recall the fact that the infecting agent resides in the secretions, and that in their transference the danger lies. Therefore, upon leaving the patient's room the careful washing of the face and hands and the brushing of the shoes are positively essential. The patient should have separate dishes and eating utensils. Better than serving meals on dishes to be brought into the room is the practice of putting the food into special ones from the patient's apartment, placed just outside the door, care being taken not to touch them. Nothing should be allowed to leave the room until after thorough cleansing and sterilization. Especial care as to excreta, soiled dressings, and handkerchiefs is imperative. For the latter pieces of gauze should be used, and burned like everything else of negligible value. To prevent the carrying of infectious material by insects, the room or apartment should be thoroughly screened. It is hardly necessary to mention that a large, airy, well-ventilated room is to be preferred, but it should be one free of draughts. After the patient has recovered and is ready to leave the apartment, provision should be made for its immediate and thorough cleansing and fumigation. My own feeling is that a painstaking washing of the walls, floors, furniture and utensils with soap and water, and the steaming or sunning of mattresses and linen will be all that is required. Where possible, the refinishing of the walls, floors and furniture will add to the feeling of security.

Medical and Hygienic Management. A diagnosis of scarlet fever having been made and the patient isolated, the first thing of importance is to put the patient to bed, even though the attack be of the mildest form. It is too common an experience to see children upon whom the rash is still visible up and about the house. The patient should be kept in bed for at least four weeks; especially is this advisable in the smaller children. The principal reason for this is the avoid-

ance of draughts and changes of temperature, thus reducing to a minimum, in conjunction with our other care, the patient's liability to nephritis, rhinitis, and other complications. The temperature of the room is of vital importance at all stages of the disease. I have found that the severer acute cases, and particularly the septic ones with hyperpyrexia, restlessness and delirium, thrive best when the temperature is maintained at between 55° or 60° F. (12.7° or 15.5° C.). The milder acute cases, as well as the convalescents, do well in a temperature of 68° to 70° F. (20° to 21.1° C.). When the patient is once out of bed he may, if not too weak, be allowed out-of-doors on warm, quiet, sunny days.

The isolation of the patient having been arranged, and the length of the stay in bed having been thoroughly impressed upon the family and attendants, with the understanding that it is to be absolute, the general care of the patient must receive our next attention. Warm bed-apparel should be the unvarying rule. A flannel shirt will prevent chilling of the shoulders and chest, the parts least protected by the bed-coverings. Careful attention to the skin should be given from the very first. A daily sponge or bath with soap and tepid water is positively called for, inasmuch as it contributes to bodily comfort, assists in elimination, and helps restore the skin to a normal, healthy condition. Inunctions are of no value as a routine measure, except when used to prevent the scattering of the exfoliating skin.

Other matters of importance pertaining to the general hygiene of the patient are careful attention to the kidneys and bowels. The menace of nephritis always should be kept in mind. The daily measuring and recording of the urinary output, and the chemical analysis every second day are absolutely imperative. If there be any albumin, a trace even, a daily analysis, microscopic and chemic, should be done until the analysis is normal again.

At least one good movement of the bowels should be assured every day. For smaller children castor oil, or pulvis glycyrrhiza compound, will do good; for older children and adults nothing is better than salines. An occasional colonic irrigation, say twice a week in mild or convalescent cases, will do much by keeping clean the lower bowel.

The diet in scarlet fever always should receive the closest attention. As vomiting in most cases is quite persistent during the first twenty-four hours, it will do no harm, and even be helpful, to withhold all nourishment during that period. Thereafter, during the acute stage, a pure milk diet is the ideal one. It is my practice to have the patient given food at three-hour intervals; as much milk as the patient may desire. The patient, however, is not to be awakened for food or medicine. Meat-broths are not desirable if milk can be taken. In addition to the milk, a daily feeding of some fresh fruit juice, well diluted with water, should be given one hour before the regular nourishment. Water should be given freely at regular intervals, and between at the patient's request. There is no objection to the use of good carbonated waters, which oftentimes will be found more satisfying to adults than plain water.

In mild cases and in those of moderate severity no difficulty will be encountered in maintaining the patient's strength on a strict milk diet. The average case I keep on milk and fresh fruit juices until the temperature has been normal for from seven to ten days. Then, if the kidneys be in good condition, secreting a normal quantity with negative findings, the diet may be increased by the cautious addition of strained oatmeal, the pulp of stewed fruits, toast, bread and butter, and puddings of a simple nature. Weak tea and coffee may be allowed those accustomed to their use. This dietary should prove sufficient until the patient is safely past the fourth week, the stage of the disease when nephritis is most likely to show itself. After this various vegetables, fish, and the white meat of chicken may be permitted. In septic cases or those in which the fever is unusually protracted, or the patient further weakened by complications, a resort to a diet other than milk may be necessary at an earlier time than in ordinary cases. In cases of persistent vomiting rectal feeding may be necessary, using peptonized milk, beef-juice and whisky in saline solution. Sore throat may be of such a degree as to render swallowing difficult and make nasal feeding necessary. It is rarely, however, that this condition cannot be met successfully by the exercise of patience or tact on the part of the parent or nurse.

In considering the medical treatment of scarlet fever it must be remembered that the exact etiology of the disease is unknown, and that specific treatment by sera or vaccine is out of the question. Nor have we at our command any drug or other method of treatment which is in any way specific.

There are two cardinal symptoms, or conditions, which underlie our diagnosis of scarlet fever, the rash and the angina. The rash rarely requires treatment. A bright, well-developed rash denotes good heart action, and is to be desired. Should the rash be scant and not of good color, the use of external measures, such as the hot pack or wrapping the patient in blankets with hot-water bottles, is called for. This is true even when the temperature is high, because the resulting increase of skin elimination will bring with it a drop in temperature and make the patient less restless.

The sore throat of scarlet fever needs attention entirely according to its severity. In smaller children the milder degrees of sore throat will do best if left alone. In the older ones, those able to gargle, the use of a solution of normal saline or of the liquor antisepticus alkalinus of the National Formulary three times daily is desirable. Local treatment of the throat by irrigating, syringing or swabbing is to be condemned, except under certain conditions. Where the patient is an adult or a child sufficiently tractable to co-operate, irrigations of normal saline, as hot as can be borne with comfort, may be allowed. Again, when ulceration occurs, the gentle cleansing with one-half strength hydrogen peroxid and the subsequent application of one-half saturated solution of potassium chlorate to the affected parts only are indicated. But before permitting this the physician should be sure that the nurse in charge is a dependable person, who thoroughly understands that gentleness should be the outstanding feature of the operation. In certain prolonged cases, however, the application of a 5 per cent. copper-sulphate solution, instead of the potassium chlorate, will be necessary, but these instances will be rare. Local applications should be made twice daily.

The mildness of throat symptoms at the outset should not lead us to neglect the daily routine examination of the mouth. The condition of the throat in the acute stage of almost every

case is a fair index to the patient's general condition. Furthermore, by this daily inspection various forms of stomatitis, tonsillitis, diphtheria, and the Koplik spots of measles may be recognized early and promptly treated.

The only medicinal remedy that I use as a routine during the febrile stage of scarlet fever is citrate of potassium, given every two hours in full doses to adults, and in corresponding doses to children, 1 grain to every year of age, in an abundance of water. When the patient's temperature has been normal for four or five days Basham's mixture is substituted for the potassium citrate. As a roborant, when the patient is on full diet, the syrup of iodid of iron, in combination with the syrup of hypophosphites, is preferred.

The complications of scarlet fever call for prompt and effective treatment. For purposes of convenience we may divide them into those that occur during the stage of fever and those that occur during convalescence.

Early Complications. The treatment of *scarlatinal sore throat* has already been described in detail. In addition to a pharyngitis, there may be an abscess, either a true quinsy or a retropharyngeal abscess. These are comparatively rare, especially the latter. Both must be treated in the usual manner by prompt incision and ordinary after-treatment.

A minor complication often met with is *cracking of the lips,* and for the relief of this troublesome, but not serious condition, I have found compresses of camphor-water more efficacious than ointments. Oftentimes the splinting of the child's arms will be necessary to prevent picking, and thereby infecting the lips.

Ulcers of the aphthous variety are sometimes encountered on the tongue of cases in which the temperature, for unexplained reasons, has failed to come to normal, remaining around 100° and 101° F. (37.7° and 38.3° C.). Potassium chlorate, as previously mentioned for other mouth conditions, will prove a specific for this aphthous stomatitis.

The *nose* in scarlet fever may be a source of great trouble, and rhinorrhea is one of the most intractable complications of the disease. Its existence lengthens the infectious period and thereby prolongs quarantine. Conservative treatment, as with the throat, is the best. To prevent the formation of

crusts and thereby to facilitate drainage I have found a combination of menthol, 2 grains (0.13 Gm.); camphor, 3 grains (0.195 Gm.); eucalyptol, 1 fluidram (3.75 mils), and liquid albolene to make 1 fluidounce (30 mils), to be of advantage. Five drops of this should be instilled into each nostril three times daily. Argyrol instillations I have tried, but with indifferent results.

The *eye complications* met with in scarlet fever are conjunctivitis, blepharitis and ulcerative keratitis, and with the exception of the last named, they are rarely serious. They require the same treatment as when due to other causes.

Laryngeal complications of scarlet fever are infrequent, although membranous or ulcerative laryngitis may occur. In some instances intubation or tracheotomy may be required, and it will be impossible to differentiate the condition from the laryngitis of diphtheria, except by repeated culture and careful observation.

In the milder cases, *pyrexia* requires no treatment other than the ice-bag to the head. In the severer forms, tepid sponges every three or four hours should be employed, with a colonic irrigation once daily, preferably in the early evening. If delirium and restlessness persist, the use of bromids in doses of 10 grains (0.65 Gm.) every two hours for a child 10 or 12 years of age is indicated, and will usually prove sufficient. In severe cases of delirium, chloral hydrate should be given in combination with the above, 5 grains (0.325 Gm.) every two hours for three or four doses. Great care always should be exercised in giving chloral, and I recommend it only in those cases in which the restlessness is most pronounced. Other drugs that have proven efficient at times are veronal and paraldehyd. Opium I rarely use in any form.

We must distinguish between the early *febrile albuminuria* and the true nephritis of scarlet fever. Simple albuminuria occurs earlier in the course of the onset, and disappears, as a rule, with the fever. An early true nephritis, however, must be reckoned among the possibilities, and must not be treated as a simple albuminuria. The continuance of a milk diet and the use of potassium citrate with water given freely will, as a rule, be the only treatment necessary for a simple febrile albuminuria.

Arthritis occurs in 3 per cent. of all scarlet fever cases, the joints most commonly affected being those of the wrist, hand and fingers. The larger joints are occasionally, but very rarely, arthritic; and in these suppuration may occur, although it does not occur in the smaller joints. The treatment is the same as that for any other inflamed joint condition of a non-surgical nature. The use of aspirin or the salicylates in alkaline solution will be sufficient internal medication. The joint affected must be put absolutely at rest by splints; local applications, such as 10 per cent. ichthyol ointment or lead-water and laudanum, may be employed. Constant moistening of the dressings with a saturated solution of magnesium sulphate often relieves the pain and inflammatory symptoms.

Ear complications in scarlet fever are not only at times grave, but have much to do with the actual duration of the infectious period of a given case. Acute otitis media may occur at any time in the course of the disease, and, therefore, a careful examination of the drum-membrane should be made a part of the routine. It commonly occurs in cases in which there has been marked sore throat or rhinorrhea, but it may occur with the mildest of cases and without warning. Oftentimes the discharge is the first and only evidence. As soon as any symptoms indicate ear trouble a thorough examination should be made, and, if the drum-membrane should be found red or bulging, free incision is called for at once. This should be done in the lower posterior quadrant. There can be no doubt that by temporizing with middle-ear disease we invite graver ear complications, such as chronic otitis media, mastoiditis, thrombosis and cerebral abscess. After incision of the drum-membrane the relief of all symptoms is immediate, and further local treatment is rarely necessary during the further course of the acute stage. External cleansing and keeping the canal clear to maintain free drainage are all that is needed. Routine irrigations I do not advise. However, should local treatment at any further time be indicated, it should be done by the doctor only, and never without the use of a head-mirror and speculum.

Mastoiditis may supervene upon otitis media. With the application of external heat and extra attention to the procuring of drainage through the ear, operation may be avoided in

some instances. Should the redness and tenderness behind the ear become marked or the patient show signs of severe systemic disturbance, and especially if fluctuation be present, excision of the mastoid cells should be done. Rarely is further interference justifiable at this stage of the disease. Should the condition become chronic, radical and more complete operation may be done, always by a competent surgeon, after the purely scarlatinal part of the disease has spent itself.

Of the late complications, or those occurring when convalescence should be well under way, *postscarlatinal nephritis* is the gravest and most important. Attention already has been drawn to the frequency of febrile albuminuria during scarlet fever, and the possibility of an early nephritis.

The nephritis of scarlet fever is characterized especially by changes in the glomeruli, although the pathologic process affects the structures of the kidneys in general. The occurrence of casts or a diminution in the daily quantity of urine should at once lead to very active measures of treatment. Edema is rarely encountered where there has been a systematic watching of the daily urinary findings. It is only in the neglected cases that this symptom draws the physician's attention to the underlying nephritis. Some degree of nephritis is found in about 10 per cent. of all cases of scarlet fever. It rarely becomes chronic.

The treatment of nephritis following scarlet fever must be prompt and vigorous. It must include: (1) Restriction of the diet to milk and the administration of water in considerable quantities. Later in the disease, say after three or four weeks, when there are present anemia and emaciation, even though albumin may persist in the urine, the diet may be increased to advantage by the addition of eggs, cereals, bread and butter, stewed fruits and puddings. (2) Free elimination by the bowels. For this purpose I employ magnesium sulphate or pulv. jalapæ comp. for adults and larger children, and oleum ricini for children 4 years old and under. (3) The use of diuretics. Potassium citrate alone or with sp. ætheris nitrosi and liq. ammonii acetatis is given as a routine. In the later stages of nephritis, when anemia is present, Basham's mixture is substituted. Other diuretics that might be used are caffein and diuretin. (4) The hot pack. This should be

used at intervals varying in frequency from once daily to every four hours, and should be continued until examinations show the urine to be negative on four successive days. After this the pack should be withdrawn gradually, and the diet correspondingly increased. I consider the hot pack as our most important means of combating nephritis, inasmuch as the increased skin elimination lifts the bulk of the burden from the crippled kidneys.

Under such vigorous treatment uremia should not supervene. However, if this should develop, purgation with Ol. tiglii or elaterium, and venesection followed by salt solution intravenously should be the measures immediately adopted. The use of dry cups over the kidneys and hot saline by the bowel is also indicated. Whether poultices do any good I cannot say positively, but they do no harm, and are, therefore, used. Oftentimes their use brings from the patient an expression of comfort. They should be composed of flaxseed and powdered digitalis-leaves, 4 parts to 1, or flaxseed and mustard, 16 parts to 1. They should be large enough to cover the entire lumbar region and applied warm.

For *adenitis,* usually of the cervical variety, the best treatment is heat, applied as hot fomentations or by means of poultices. Should softening occur, free incision and drainage are indicated. The fear on the part of some that heat from poultices favors suppuration in the adenitis of the acute stage of the disease has not been justified by my experience. Local medicinal applications I have not found of advantage in shortening an attack of adenitis or in preventing suppuration. The possibility of the glandular discharges containing the infecting agent of scarlet fever and the individual thereby becoming a carrier should always be borne in mind.

Chest complications in scarlet fever are rare. Those affecting the heart are endocarditis, pericarditis and myocarditis. The lung complications are bronchitis, bronchopneumonia, lobar pneumonia, pleurisy and empyema. The treatment required is the same as when due to other causes.

Reinfections or relapses in scarlet fever occur oftener than is generally supposed. By a relapse we mean the redevelopment of the disease in an individual before complete recovery from the first attack. Some authorities state that the severity

of a relapse is in inverse proportion to that of the first attack. They usually occur about the middle of the fourth week. The symptoms are like those of the first attack, vomiting, sore throat, fever and a rash, followed by desquamation. As a rule, relapses are mild and end in recovery. The treatment is the same as for the initial attack.

Second attacks of scarlet fever, those occurring after full recovery, may occur also, the period of time between the attacks varying from six weeks to several years.

Scarlet fever seems to prepare a fertile field for the development of other infectious diseases, especially for diphtheria, measles and varicella. The daily routine of inspecting the throat, as a means of early recognition and prompt treatment, has been mentioned as a necessary procedure in all cases of scarlet fever. In the severe anginose cases of scarlet fever, even where the chances of diphtheria being present as a coincident infection are not very strong, the giving of a curative dose of diphtheria antitoxin is indicated. It can do no harm.

Other conditions that may complicate scarlet fever, though rarely, are meningitis, jaundice, peritonitis, eczema, vaginitis and postfebrile mania. They require no special treatment.

And lastly, there must be included in the treatment of scarlet fever the determination of the time when the patient is fit for release from quarantine; when, as near as we can judge, he ceases to possess the power to infect. Because of its bearing upon the general public health there is nothing which calls for greater care than this decision. There are no certain means by which we can arrive at such a conclusion. "Return cases" occur in about 2 per cent. of all discharges from hospitals, according to statistics. My own methods in this matter, based upon experience only, approximately are as follows:

All adults and larger children, never the subject of complications, may be discharged at any time after a detention of thirty-five days dating from onset of the illness.

Mild cases in smaller children, not the subjects of complications, will require a minimum detention period of forty-two days.

Cases with rhinorrhea should be detained three months, and in every instance should be cultured for diphtheria bacilli.

Otorrhea calls for detention of from three to four months. "A running ear" should be kept for four months; a moist ear, in which but a few drops show every day or so, may be released in three months.

Cases with acutely inflamed glands should not be discharged until all signs of inflammation shall have disappeared. Suppurating glands must have healed perfectly before the release of the patient is permitted.

Desquamation in itself probably does no harm in any stage of the disease, and certainly none after the end of the fourth week. Because, however, of the universal belief in the infectiousness of the scales of the skin, it is my custom to see that all cases are free from them before being released. In most all cases desquamation is complete before the end of the sixth week.

At the time of the patient's discharge he should be given a thorough and vigorous cleansing bath of soap and water. This should include the scrubbing of the head and the cleansing of the nose and the ears. The mouth should be made clean with a mild antiseptic gargle, after attention to teeth and gums by a dental surgeon.

The apartments occupied by the patient should be cleaned and fumigated in the manner already mentioned. (See p. 169.)

And finally, in spite of the most careful observation of the patient from the beginning of illness to the end of quarantine, and in spite of the most painstaking attention to everything connected with the treatment, the greatest danger of spreading the disease will be the patient himself.

MEASLES.

Measles is an acute contagious disease characterized by a peculiar eruption and a catarrhal condition of the upper air-passages and bronchi.

The etiology of measles is unknown. It partakes of all the characteristics of a disease caused by a micro-organism, but the specific causative factor has not been isolated.

The mode of transference of the disease is doubtless most often by direct contact. The most infectious period of the disease is the early one, when the discharges from the nose

and eyes are most marked, and before or just when the rash is beginning to appear. It may be possible that the disease could be carried by a third person or object by the transference of small particles of infectious material, but this is, I think, quite rare, owing to the fact that the virus is very vulnerable and short-lived. Apparently measles is not spread by water or by food, nor, contrary to the old belief, are the desquamating particles of the skin infectious.

Measles may occur at all ages of life, but under ordinary conditions it is essentially a disease of childhood. It is probably the most widely disseminated of all the infectious diseases, and in epidemics attacks persons of all ages who are not immune because of a previous attack. Immunity is conferred by a previous attack in almost all cases.

The seriousness of measles in epidemics among uncivilized peoples has always been recognized, but ordinarily it is considered too lightly. Bearing in mind the frequency and the severity of its gravest complication—bronchopneumonia—it is one of the most fatal and serious of all the contagious diseases.

The period of incubation of measles varies between seven and eighteen days; on an average it is ten days after exposure before catarrhal symptoms begin, and fourteen days before the appearance of the rash. The symptomatology of measles is a characteristic one. The period of invasion is characterized by fever tending toward remissions to, or nearly to, the normal temperature range.

We find also coryza, and, in fact, a catarrhal condition of the nose, eyes and bronchi, with slight cough. The eyes are congested and sensitive to light, and there is a free lachrymal discharge. Pathognomonic in this stage is the appearance of Koplik's spots. These are described as minute, bluish-white spots, often with a red areola, occurring upon the mucous membrane of the mouth as early as the first day of the invasion. When diffuse they are very easy to recognize.

The rash usually appears upon the fourth day, though it may occur earlier or later. First noticed on the face or neck, the eruption begins as a macular type, and later becomes papular. The lesions may remain discrete or become coalesced, and the whole body is soon covered with the erup-

tion, which may, in very severe cases, become hemorrhagic. The rash and accompanying symptoms of measles may range from the mildest to the most severe conditions.

Within from two to five days after the onset and full development of the rash, it begins to fade, the other symptoms abate, and the temperature rapidly falls to normal in uncomplicated cases. As the rash fades it almost always leaves a characteristic coppery mottling or discoloration of the skin.

Measles might be confounded with scarlet fever, German measles, and the incipient stage of smallpox. Except under most unusual circumstances, the diagnosis of measles is very easy.

TREATMENT.

If uncomplicated, measles requires but little besides general care. A case of measles should be kept in a room not too cool, from 65° to 70° F. (18.3° to 21.1° C.). The patient should be well protected from the possibility of exposure to changes in temperature and draughts, and the room should be kept dark so long as photophobia and congestion of the eyes and conjunctivæ persist. The bowels must be kept open, and the diet in the febrile stage be of the lightest variety, with water given freely.

Stimulation is, as a rule, not needed, unless in those asthenic cases of measles that occasionally are met with during the course of severe epidemics. The renal function is but rarely impaired in measles. When slight febrile albuminuria occurs, the use of citrate of potassium and the free use of water practically always brings about prompt abatement of this symptom.

Should the rash of measles be not well shown, it is my practice to bring it out by the use of a hot bath, due precautions being taken that the patient does not take cold. Other local treatment is not required. Proper and sufficiently frequent cleansing of the skin aids desquamation and shortens its duration.

The eyes of the measles patient require protection and cleansing. In the mildest of cases no washing may be needed. If, however, special care be necessary, a boric acid solution

will usually be found sufficient. If this does not fully answer the purpose, instillations of 10 per cent. argyrol solution once or twice daily will hasten a cure. The lids become inflamed in the presence of free discharge, and require the application of some bland ointment, such as the ointment of the yellow oxid of mercury in the strength of 1 grain (0.065 Gm.) to the ounce (30 mils).

Should symptoms of keratitis or corneal ulcer supervene, a specialist should be consulted.

The condition of the respiratory tract should always be kept under close observation in measles. A mild catarrhal laryngitis is very common in measles, but this requires no particular treatment. The chief importance of more severe grades of laryngitis complicating measles lies in their possible confusion with a diphtheria complicating the disease. It is well known, and should always be borne in mind, that measles predisposes to diphtheria.

Bronchopneumonia as a complication of measles is of the utmost importance, as it is the chief cause of the very appreciable death-rate in this disease. Its onset may be looked for during the eruptive period, and if, when the rash fades, the lungs do not clear up and the temperature remains above normal, we should at once suspect some grave pulmonary lesion.

The treatment of bronchopneumonia and other complications, such as otitis media, in measles does not differ from that employed in children when they arise from any other cause.

The treatment of the complications of measles, and especially bronchopneumonia, is, and should really be, largely prophylactic. If due regard be given this in preserving a proper temperature of the room, with sufficient aëration, and if the patient be kept in bed until a complete curé has been effected, pulmonary complications will be rare. In patients who are weakly and the possible subjects of tuberculosis in any form, care should be exercised for a number of months after an attack of measles, in order to avoid such possible after-effects.

GERMAN MEASLES.

German measles, or rubella, is a specific and infectious fever, of short duration, attended by few constitutional symptoms, and characterized by a papular or macular eruption.

As a disease German measles is a distinct entity, in spite of the prolonged discussions that have arisen as to its true nature. It is in nowise connected with scarlet fever or measles; it is neither of these diseases in modified form, nor is it a hybrid between the two.

The contagiousness of German measles is much less than that of measles, and for that reason widespread epidemics are seldom seen. It occurs with the greatest frequency during the spring and early summer months. From the experience of most contagious hospitals, it would seem that the disease occurs with exceptional frequency in young adults. It is seldom met with, however, in persons over 30 years of age. Sex appears to be without influence upon its incidence.

The disease is most contagious when the eruption is at its height. The nature of the contagion is not known; it is most probable that this resides in the catarrhal discharges. The mode of infection is by direct contact of the sick with the well. It is rare that third parties or fomites play the rôle of carrier.

The incubation period varies from twelve days to three weeks. The average is fourteen days.

The classical symptoms of German measles are malaise, mild coryza, fever, and the rash. Swelling and tenderness of the lymphatic glands is an early symptom in most cases. Enlargement of the post cervical glands is considered of diagnostic value by many. Suppuration of the lymphatic glands is never known. As a rule, the rash and slight fever will be the only symptoms. The fever in the milder cases may remain at normal during the entire course of the disease; on the other hand, it may go as high as 104° or 105° F. (40° or 40.6° C.). A curious and important fact is that with the very high fever there is no prostration. Very rarely does the patient appear uncomfortably or seriously ill. The fever returns to normal before the complete fading of the rash.

The rash shows first on the face, and rapidly extends to the trunk and extremities. It begins as small, very slightly elevated papules, fairly discrete, and similar somewhat to those of measles. These fade quickly, and leave behind no staining or mottling of the skin. The duration varies, as a rule, from twenty-four to forty-eight hours; it may, however, remain out for so short a time as twelve hours, and, again, may last for four or five days.

Complications, sequelæ, or relapses we do not see. It is possible that second attacks do occur, but they are certainly very rare. Recovery is the unvarying rule. I have frequently noted with much interest the little disturbance caused by an attack of German measles in patients already the subjects of other diseases, such as scarlet fever and true measles.

The diagnosis is the most important feature in the handling of German measles. Failure to diagnose the disease correctly has been the source of much embarrassment to health authorities, and of great humiliation to the attending physician. The diseases from which it should be differentiated always are scarlet fever and true measles, particularly the former. Other and less important conditions with which it may be confused are rashes due to drugs, and to errors of diet.

TREATMENT.

There is no contagious disease which requires so little treatment. Rest in bed until two or three days after disappearance of the rash will be all-sufficient. In a few days more the patient may be allowed out-of-doors on quiet, sunny days. Should there be fever, a light diet should be enforced until the return of the temperature to normal. Ten days after the appearance of the eruption the patient should be considered fit for discharge.

Tropical Diseases

BY

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Tropical Diseases.

FOREWORD.

One of the most satisfactory things connected with tropical medicine is the fact that the more important diseases are preventable. Their prophylaxis is well understood. Modern medicine has probably received its greatest renown through the discoveries made in the prevention and cure of tropical diseases. The conclusive proof that the mosquito conveys filariasis, malaria and yellow fever, that beriberi is due to improper diet, that cholera and hookworm are contracted through contact with human feces, and the conclusive results that have followed the application of the indicated measures, are among the brightest pages in medical history. The lives saved already run into the hundreds of thousands, and the distress and suffering avoided goes into the millions.

The object of this section is only to give brief descriptions of a few of the more important diseases, and to afford a ready reference to those commonly found in the tropics. In these days of rapid communication residents of warm countries frequently call upon the doctor of temperate zones for medical relief, so physicians of all climes have an added incentive to acquaint themselves with the diseases of all regions.

The strides in tropical medicine are so rapid that unless the subject-matter is constantly revised and brought up to date, it is of very little practical value. While it is not primarily the purpose in this chapter to dwell upon hygiene, sanitation and prophylaxis of tropical disease, yet these factors are so intimately associated with the subject that a brief *résumé* of the modern methods in this regard will be included.

LEPROSY.

Leprosy is a chronic infectious disease caused by the *Bacillus leprœ,* and usually occurs in one of three types. When the principal disturbance consists of anatomic changes in the

nervous system, accompanied by loss of sensation and atrophy, it is referred to as anesthetic leprosy. When it is characterized by the presence of tubercular nodules in the skin and mucous membranes, it is called tubercular or hypertrophic leprosy. When it is a combination of these two types, it is called mixed leprosy.

In the struggle between man and disease there is no malady which has aroused greater universal sympathy, and, until recently, has baffled all efforts toward successful treatment. Leprosy is one of the first diseases of which we have a record. Mention is made of it in Egypt 1500 B. C. It is described in the Bible. It was common in Italy during the time of Pompey, and subsequently extended to all parts of the earth. The movement caused by the Crusades was probably responsible for the transmission of leprosy to Western Europe, and its appearance in epidemic form.

Greek writers, perhaps, have given the best of the early descriptions of leprosy. For instance, the following is from Aretæus:

"Shining tubercles of different size, dusky red or livid in color, on face, ears and extremities, together with a thickened and rugous state of the skin, a diminution or total loss of its sensibility, and a falling off of all the hair except that of the scalp. The disease is described as very slow in its progress, sometimes continuing for several years without materially altering the functions of the patient. During this continuance great deformity is generally produced. The alæ of the nose become swollen, the nostrils dilate, the lips are tumid; the external ears, especially the lobes, are enlarged and thickened and beset with tubercles; the skin of the cheek and forehead grows thick and tumid and forms large and prominent rugæ, especially over the eyes; the hair of the eyebrows, beard, pubes and axillæ falls off; the voice becomes hoarse and obscure, and the sensibility of the parts affected is obtuse or totally abolished, so that pinching or puncturing gives no uneasiness. This disfiguration of the countenance suggested the idea of the features of a satyr, or wild beast; hence the disease was, by some, called satyriasis, or by others leontiasis. As the malady proceeds the tubercles crack and ultimately ulcerate. Ulcerations also appear in the throat and nose,

which sometimes destroy the palate and septum, the nose falls, and the breath is intolerably offensive; the fingers and toes gangrene and separate joint after joint."

Leprosy increased in Europe in the days of Pompey. In the thirteenth century it became epidemic. Stern measures were enforced, and lepers were isolated in colonies. They were often required to wear special dress or to ring a bell when passing along the street. They were forbidden to drink at public fountains, or to touch children, or to eat with persons other than lepers. The church performed the funeral service over persons who were diagnosed as lepers, and they were regarded as dead. This latter provision is said to be still carried out in order to permit lepers who have non-leprous husbands or wives to marry leper inmates of colonies. The West Indies gradually became infected, probably through the Negro slave trade. The disease was carried to North America, where it has been slow in spreading. About the same time it was carried to South America, where it has flourished, especially in Brazil and Venezuela. In Africa it was first reported in the South in 1756, and is said to have been carried there by the Dutch. Since that time the disease in Africa is said to have increased mainly among the East Indian troops. In Biblical and other older writings it is more than likely that diseases described as leprosy are often some other disfiguring skin diseases which resemble it. There is much reason to believe, for instance, that frambesia exists in three stages, like syphilis, and that in the third stage the lesions and deformities are often mistaken for leprosy. Syphilis, psoriasis, tubercular ulcers, madura foot, ichthyosis and other skin affections have undoubtedly been mistaken for leprosy in the past. The diagnosis was not placed upon a scientific basis until the discovery of the bacillus by Hansen, which is now known by his name. Failure to cultivate the bacillus has probably been one of the greatest stumbling-blocks in developing a satisfactory treatment. The disease has gradually disappeared in those countries in which effective efforts have been made to isolate those suffering from the disease. It may be regarded as axiomatic that no new case of leprosy occurs in any geographic area unless there has been a human leper there before.

Many authors hold that at the present time leprosy is more particularly a disease of tropical and subtropical countries. The fact that this distribution does correspond with those latitudes does not necessarily mean that there is anything peculiar about the climate which promotes the transmission of leprosy. The fact that in most temperate countries isolation is more rigidly carried out, and that therefore there is not the same opportunity for the spread of the disease, may be the real explanation. Leprosy is very common in some cold countries, notably in Finland, Norway and Sweden. It is also reported to be common in Russia, near Riga, but there are no reliable data with regard to this point. In the United States the two States having the greatest numbers are Louisiana and Minnesota, the first being subtropical, and the other extremely cold. In Hawaii the disease spread rapidly after 1860, and strenuous efforts to stamp it out have not succeeded in greatly reducing the incidence. A search through the literature fails to show any report of the disease before 1848. In 1865 there were 230 known lepers in a population of 67,000; by 1891 there were 1500 lepers in a population of 44,000, or one in thirty. There is much reason to believe, however, that segregation of lepers has not been effectively carried out in the Hawaiian Islands. It cannot be very well charged that isolation has failed to stamp out the disease, because isolation has not been given a fair trial. Leprosy is also common in some South American countries, notably in Brazil, Venezuela, Ecuador, Argentina and Colombia.

It is more than likely that leprosy is much more common today than is generally suspected, especially in countries regarded as more or less free of the disease. It is estimated that in India, among a population of 210,000,000, there are 105,000 lepers. This gives a ratio of one to 2000. There are various estimates of the number of lepers in Japan, but a conservative figure would perhaps be 50,000. Calculating the population of Japan at 50,000,000, this would give an incidence of one to 1000. In the Philippine Islands, with a population of approximately 8,000,000, 10,000 lepers were collected between 1906 and 1914. It is not likely that there were more than 6000 cases of leprosy during any one year. This would give a ratio of one to 1400. It is usually asserted that leprosy

is more common among those of uncleanly habits, where there is squalor, dirt and poverty. The dark skinned races were thought to be especially susceptible. A rough calculation of the number of Americans who have been in the Philippines shows that the ratio among them is about one to 1400, which is the same as that for the Filipinos. A recent calculation of the number of lepers in Java shows that the incidence of the disease is apparently as great among the Europeans as among the natives. In China the disease is very common in the Southern part, and apparently rare in Northern China. In India there is a similar disproportion between the number in the North and in the South. New Caledonia is another country into which the disease was introduced in comparatively recent times. The first record of it was in 1865, and it was presumably introduced by Chinamen. There are now probably 5000 lepers in New Caledonia. In countries where public health and sanitation are exceptionally good, leprosy may occur. New Zealand, for instance, with a population of 1,000,000, is reported to have five lepers, or one in 200,000. Australia, with a population of 6,000,000, is reported to have thirty lepers, or one in 200,000. The United States, with a population of 100,000,000, has perhaps 1000 lepers, or one in 100,000.

For convenient reference the following table is submitted as a rough estimate of the proportion of lepers to the population in different countries:

Japan	1 in 1,000
Philippine Islands	1 in 1,400
India	1 in 2,000
United States	1 in 100,000
New Zealand	1 in 200,000
Australia	1 in 200,000

A small rod-like organism closely resembling the tubercle bacillus discovered by Hansen of Bergen in 1871 is very generally recognized as the cause of the disease. Many efforts have been made to cultivate the micro-organism, but so far without demonstrable success.

There is no definite information available as to the method by which the disease is transmitted. It is generally held that it may take place by inoculation, and in support of this the

case of the Hawaiian convict is usually quoted. This prisoner, who was under sentence of death, was inoculated on September 30, 1884, by Arning. Four weeks later he had rheumatoid pains and gradual painful swellings in the ulnar and median nerves. The neuritis gradually disappeared, and then a small leprous tubercle developed at the site of the inoculation. In 1887 the disease was quite manifest, and the man died six years after inoculation. However, the case is not regarded as conclusive, because the man had leprous relatives and lived in a leprous country. It is not likely that the disease can be transmitted through heredity. A most careful examination of fifty children born of leprous parents at the Culion Leper Colony did not result in the discovery of a single case of leprosy. One of these children developed leprosy at the age of 2 years. However, this child lived at the leper colony in close intimate contact with its mother, and probably contracted the disease there after birth. It is very difficult to show that leprosy is contracted through contagion. An ulcer, or the terminal effects of an ulcer, located at the junction of the cartilaginous and bony portion of the septum of the nose, in which are imbedded leprosy bacilli, is one of the most constant signs of the disease. It is possible that contagion may be given out to the surrounding air through open ulcers of this kind which contain leprosy bacilli. Glass plates that have been exposed to the breath of such persons have resulted in the finding of leprosy organisms. Later, however, considerable doubt has been thrown upon these experiments, because it not infrequently happens that an acid-fast bacillus can be found in the greasy exudates of the face which may not be leprosy bacilli. At times cases of leprosy occur in communities where there is apparently no connection with a previous case. Such an instance occurred a few years ago, in Indiana, in an old negro woman who had never been out of the State; the diagnosis in this instance was well authenticated, and it is generally reported that there are no other cases of leprosy in Indiana. During the present year (1917) another case has been reported, but the infection is believed to have taken place outside of the State. However, it is very difficult to be dogmatic as to the absence of leprosy in Indiana, and more detailed information with regard to the

case might show that the woman had been in contact with a leper. It is also frequently stated that washerwomen are more susceptible to leprosy than others. Statistics of the occupations of 10,000 lepers in the Philippines show that there is no special disproportion among the various occupations.

There is considerable evidence that infection may take place through food. There is no record of leprosy having been contracted on a leper colony in which food contamination could be clearly excluded.

Sexual intercourse also seems to be frequently responsible, but that it is not the only mode of infection is clearly shown by the fact that 2-year-old children have been known to acquire the disease.

As a rule, doctors and nurses in leper colonies do not contract the disease, although there are a number of notable exceptions. One of the best known cases is that of Father Damien, who became a leper at Molokai, in the Hawaiian Islands. There is, likewise, the case of Father Boblioli, at New Orleans, and more recently (1915), the case of the Spanish priest, who contracted the disease after having served as chaplain at the Culion Leper Colony for more than two years. An investigation in the Philippine Islands showed considerable evidence that leprosy may be regarded as a house disease. In enforcing the law for the segregation of lepers it has been customary there to examine once a year the inmates of houses from which lepers have been taken. Exact statistics are not available, but a fair percentage of incipient cases of leprosy have been detected in the Philippines in this way during the past few years. Whether the disease was contracted by the contacts through prolonged intimate association with the leper who was removed, or whether it may have been conveyed by the means of bedbugs or other insects, is a much-mooted question. There are many hundreds of instances on record in which either the husband or the wife was a leper and full marital relations were maintained over a period of more than ten years, yet no infection took place. Kitasato gives the following table[1] of infections:

Children of lepers	7.05%
Matrimonial infections	8.8%
Brother and sister	4.2%

Leprosy bacilli have frequently been found in bedbugs that have fed on leprous persons, but it has not been possible satisfactorily to demonstrate that these bugs eject the organism when they bite at a later time. It has been suggested that the fecal discharges of the bedbug or other insect may be deposited on the skin, and through scratching or otherwise the infection may find its way into the body through the wound made by the insect or by way of some other abrasion.

There is a remarkable regularity of sex incidence in the disease. Regardless of what part of the world leprosy is found, there are practically always two males for every female leper. In other words, they generally go by thirds. In any given community a third of the lepers are women.

Usually the incubation period is calculated in years. The lowest known reliable record is that of an 18-months-old infant. There have been cases reported up to twenty-seven years after the last known exposure. Two or three years, however, may be regarded as a fair average incubation period.

The study of leprosy has been very much retarded, owing to the fact that no animal is known to contract the disease, and also by the difficulty in cultivating the leprosy organism. Much work has been done in recent years in cultivating the micro-organism, and the general opinion seems to prevail that leprosy bacilli can be grown in the form of a streptothrix, which later may become the characteristic rod of leprosy. It is quite noteworthy that many persons trace the first symptoms of leprosy to a prolonged exposure to salt water. This is quite a common belief among many of the islanders of the Pacific. Faint skin eruptions in leprosy often become very prominent after a hot bath.

Leprosy bacilli have also been found in the probosces of flies and upon their feet. It is quite conceivable that they might infect food or open wounds of non-leprous persons. Scabies is a skin disease which is very frequently associated with leprosy, but whether it has any direct connection with leprosy has not been proved. It may be due to the fact that scabies is particularly common among lepers owing to their uncleanly habits. It is a frequent custom among Latins and races of the Pacific not to take baths when they are ill or have a fever. For this reason some lepers do not bathe for

years, and a disease like scabies may thrive under these circumstances. The scabies itch-mite is at times undoubtedly infected with leprosy bacilli, and owing to its burrowing habits, it is quite conceivable that it might transmit the disease to others.

With regard to transmission, the whole question may be summed up as follows: There are no reliable data as to the manner in which leprosy is conveyed. So far no tenable hypothesis has been presented. Most of the evidence available shows that the disease usually occurs after prolonged intimate contact with a leper. This usually means sleeping in the same bed or in small rooms with a leper for periods of weeks or months, or through marital relations, or through the close relationship which exists between a child and its leprous mother. Whether the transmission is due to expired air, to the facilities given for insect transmission, to direct or indirect contact, to infected food, or to other relationship associated with close living conditions, is not known.

The mode of entrance of the bacillus of leprosy into the human host is unknown. The respiratory passages, and especially the mucous membrane of the anterior part of the septum, have been under strong suspicion. There is much reason to believe that at times the bacillus does enter through the alimentary canal or through the generative organs. Insect transmission, or direct contamination through the skin, also seems possible. The nature of the initial lesion, however, is unknown. The bacilli multiply enormously after they have once entered the body. Large numbers may frequently be found in the hair follicles and in the deeper-seated sections of the sweat tubules. There is no general agreement as to the first pathologic lesion. Recent observations indicate that in bone invasions the bacilli may be imbedded in a fat-like substance. All leprous lesions usually show enormous numbers of bacilli, enclosed in plasma cells, about the exact nature of which there is much dispute. These cells, containing large numbers of bacilli, are very characteristic of leprosy, and are generally referred to as "lepra cells." Often, however, there are large numbers of bacilli imbedded in a mucus-like substance. Sometimes typical giant cells, known as Langhan's cells, are seen. Ordinarily the bacilli do not invade the sur-

face of the epithelia nor the layer of the cutis directly below the epidermis. A typical leproma shows superficial epithelium, normal in appearance, except for the absence of an interpapillary process. Below the epithelium there is a layer of connective tissue, usually free from bacilli, and under this is found the typical lesion, composed of lepra cells, plasma cells and connective-tissue cells. The walls of the blood-vessels are thickened and infiltrated, sometimes to the extent of complete obliteration. In the lymph-spaces the globi of the older writers filled with bacilli are found. The attacks of leprous fever, which occur so regularly, may be explained by the dissemination of the bacilli throughout the body by the blood-stream, the germs being enclosed in large mononuclear leucocytes. Lesions may remain stationary for years, and retrogression often takes place, either spontaneously or as the result of treatment. It is assumed that the leproma press on the nerves, thus causing degeneration of the neurilemma, and, later, distintegration of the arteries and destruction of the nerve fibers. Some observers state that, not only are the peripheral nerves affected, but the bacilli attack the anterior cornua of the spinal cord. Statistics show that about 50 per cent. of lepers who have been examined are positive to the Wassermann reaction, but as frambesia and syphilis occur in so many of the lepers who have been examined, it cannot be stated that the Wassermann reaction is necessarily associated with leprosy. Efforts to recover the bacilli in the blood-stream have been very disappointing, and there is considerable doubt as to the reliableness of the reports which claim success in this respect.

The leproma is usually situated in the cutis, covered by the epidermis, but it may lie in the subcutaneous tissue, in which case it does not form a tubercle. These nodules, or tubercles, are yellowish white in color, firm in consistence, and, if squeezed, usually a little clear fluid is exuded. The sweat, sebaceous glands, and hair follicles in the infected regions are usually atrophied. The macules consist of a round cell infiltration, with few large cells, and often are free from bacilli. The spots which were anesthetic during life are usually converted largely into fibrous connective tissue, and likewise the glands and hairs in them atrophy and tend to

disapppear. The liver is usually enlarged and contains infiltrated leprous material. The ovaries and testes often show infiltration and fibrosis of the interstitial tissue, which destroys the secretory elements and causes the sterility which is usually so marked among lepers. The lymphatic glands are prone to become large, infiltrated, and filled with bacilli, especially those of the femoral regions. Nephritis and leprous infiltration are usual. There is practically always marked infiltration of the spleen, and lepra bacilli can be recovered from this organ. The nerves most markedly affected are the ulnar, the median, the peroneal and the posterior tibial. They are usually much thickened, and are the seat of a fusiform reddish gray swelling due to the presence of leprous tissue among the fibers. The spinal cord often shows posterior sclerosis and meningitis. Periarteritis and endarteritis are common. Caries, necrosis, and absorption of the bones are frequently seen. Trophic changes in the joints and perforating ulcers of the plantar region are very common in the nerve form of the disease; likewise atrophy of the interosseous muscles, the thenar and hypothenar eminences. There has been considerable difference of opinion as to whether the pulmonitis so frequently found in leprosy is due to the tubercle or to the leprosy bacilli. In more careful studies made recently it has been shown that often there is an uncomplicated leprous infiltration of the lungs. Tuberculosis of the lungs in lepers is common.

Clinical Forms (Tubercular, Nodular, or Hypertrophic). The first sign of leprosy observed is usually a sharply defined and often hyperesthetic erythema. In a small percentage of cases the earliest evidence of the disease is the appearance of small areas of anesthesia without any noticeable alteration of the skin. In practically all cases, however, when the foregoing symptoms are due to leprosy, a careful inspection of the nose will show a small area of infiltration, and often an ulcer upon the septum of the nose located at the junction of the cartilaginous and osseous portions. Even when there are no macroscopic changes, scarification of this region and an examination of the exuding fluid will show typical leprosy bacilli. In a great majority of cases of tubercular and anesthetic leprosy macules soon appear. These may vary in size

from a centimeter to areas which cover a large part of the face, or even the back. During this stage the disease progresses very slowly, as a rule. The patient often gives a history of trying various skin applications for a period of one or two years without obtaining relief. The characteristic nodules appear at periods varying from a few months to a number of years, after the first macules or infiltrations into the ears, nose, or other areas. The face is probably affected more frequently than any other part of the body. The nodules may vary in size from that of a pea to that of a walnut, or sometimes larger. These swellings often cause deformities which cause the face to resemble that of a lion, giving rise to a condition commonly referred to as the leonine facies. Sometimes the body is so studded with tubercles as to resemble masses of small red potatoes growing out everywhere. Macules or tubercles probably do not occur in the scalp, a point of much diagnostic importance, according to Hopkins.[2] Macules with elevated edges are very common on the back, and these lesions often clear up in the center and spread along the margins. The skin which has been affected by the disease usually becomes atrophic and white. The so-called typical white spots of leprosy are, however, seldom connected with the macules. They generally occur in the anesthetic form of the disease, and do not seem to be associated with inflammatory reaction. The macules often progress to crust formations resembling psoriasis. Occasionally a skin lesion resembling a typical birth-mark, or a port wine mark, is noted as one of the earliest signs in leprosy. This may occur on the face, the forearm, arms or legs. Repeated microscopic examinations from scrapings made from these lesions fail to reveal tubercle bacilli, and in several cases observed it was impossible to find tubercle bacilli in scrapings taken from the septum of the nose. After these cases were kept under observation for a period of about six months it was possible to demonstrate leprosy bacilli in the lesions and coincidently in the nose. Ulceration is not common in uncomplicated tubercular cases, although at times the tubercles break down and ulcerate. Ulcers, and especially perforating ulcers of the feet, seldom occur without it being possible to demonstrate anesthetic areas. Upon slight injury tubercles may undergo

ulceration. The tubercles are usually reddish brown in appearance. The loss of the outer half of the eyebrows occurs in fully half of the cases of tubercular leprosy.

Infiltrations without distinct nodular formation are frequently among the earliest symptoms of the disease. These are generally first shown by the swelling of the lobes of the ears and alæ nasi. Often there is suffusion of the face, which gives the appearance often noticed in photographs of persons which are slightly out of focus. Infiltrations of the larynx are also common and provoke huskiness of the voice. Infiltration into the lungs frequently causes symptoms of pneumonitis. There may be large leprous granulomata located in almost any portion of the body except the scalp. Eye lesions are very common among the lepers of some countries. In Louisiana, for instance, they are most severe, and cause suffering which it is most difficult to relieve. Among the lepers of the Philippines this is a symptom about which there is seldom complaint. Blindness, usually due to infiltrations and sometimes to ulcerations of the cornea, occurs in a small percentage of cases. Infiltrations in the nose are common and cause troublesome blocking, the tissue often breaking down and the nasal discharges containing lepra bacilli. Fever is seldom noted as one of the first symptoms in the disease. It is only after the lesions become more pronounced that attacks of leprous fever are noted. These usually have a sudden onset and fall by lysis in the course of three or four days. These febrile attacks may occur at intervals of weeks or months. No characteristic changes are in the blood.

Anesthetic Leprosy. In this form of the disease the infiltration is principally into the nerves. The onset is characterized by shooting pains, especially in the ulnar and peroneal nerves, and this may be soon followed by flushing of the face, glossy skin, and twitching of the muscles. Macular eruption often accompanies the anesthetic form of the disease. By some authorities the macular eruption is regarded as an infiltration; by others, it is attributable to tissue changes in the area supplied by the affected nerve. After the infiltration has persisted for some time the nerves can usually be felt as stiff, hardened cords, the ulnar, brachial and peroneal nerves often being readily palpable.

The macules in anesthetic leprosy are usually the same as in the tubercular variety of the disease, except that they occur with great frequency. Large areas of skin are often found to be erythematous. Erythema, or a faint eruption, may be greatly intensified by heat or other irritant, so that a diagnosis which otherwise may be obscure often becomes obvious after a hot bath. The macules commonly resemble the ringworm eruptions so frequently seen among natives in tropical countries, and these eruptions frequently disappear, especially upon the application of ordinary skin lotions. The macules are usually clear in the centers and spread from the margins, and may coalesce with neighboring macules. Areas that may involve a square foot of skin surface may result. A small erythematous patch which develops into a bright pink macule on a clear skin, and fails to heal with ordinary treatment, should be regarded with considerable suspicion. Scrapings from the septum of the nose may reveal leprosy bacilli and show the true nature of the disease. The implication of the nerves gradually becomes more serious. In those of a low order of intelligence the anesthesia often does not attract attention until the patient or some one else notices that they are insensible to burns or that they have no sensation about the feet. It often happens that the patient is burned with lighted cigars or cigarettes, or otherwise, without being aware of it. Anesthesia of the feet often manifests itself by the patient being unable to state whether he is wearing footwear. When blindfolded and barefooted he is usually unable to say whether he is stepping on stone or wood or earth. But long before these symptoms appear careful examination would reveal small anesthetic patches on the back or in the areas supplied by the ulnar, median, peritoneal, or other nerves. The ulnar side of the hand and the outer side of the foot are nearly always the first to be involved. As the nerve destruction goes on the reparative process stops, slight injuries about the extremities fail to heal, and gradually slow ulceration or absorption takes place, until the fingers or toes are completely gone. Owing to the lack of repair, lepers who play musical string instruments, have been known literally to wear their fingers away. Perforating ulcers of the feet in anesthetic cases are also quite common. These are most difficult to heal,

and usually occur at the base of the first metatarsal bone. Ulcers over the anterior surface of the tibia are also common. In anesthetic leprosy the eyebrows are seldom affected. Absorption of tissue between and in the vicinity of cartilage is likely to occur. This frequently produces considerable distortion of the nose, and deep grooves are often seen between the cartilaginous joints of the nose. Atrophy of the interosseous and thenar muscles and contractions of the thumb and fingers are also common. The "main en griff" is very characteristic in leprosy (see Fig. 3). The finger joints are ankylosed. In the hands the area corresponding to the distribution of the ulnar nerve is first affected. In right-handed persons the little finger of the right hand is usually the first to suffer. In areas corresponding to the distribution of the affected nerve, sensation may be wholly absent or greatly impaired. In the back distinction sometimes between two needle points cannot be made at a distance of 2 inches (5.08 cm.) or more. There is no uniformity between the lesions or nerves affected on the two sides of the body. Sometimes there is extreme lacrymation in one eye and none in the other. One side may have marked skin lesions or anesthesia and the other be quite free. The septum of the nose is perhaps even more frequently affected in the anesthetic form than in the tubercular. Absorption of bone frequently takes place, and through muscular contractions many odd deformities are produced, especially in the extremities. At times it appears as if the fingers had only one joint, the second and third joints having been absorbed, the joint with the finger-nail being close to the carpal bones. The duration and progress of anesthetic leprosy is usually much slower and more prolonged than is the rule in the nodular variety. The only symptom for many years may be a slight contraction with ankylosis of the little finger. Leprous fever in the anesthetic form seldom occurs, and is never so severe in cases in which the anesthetic symptoms predominate as in the nodular variety.

Mixed Leprosy. Mixed leprosy is simply the presence in the same person of both forms of the disease heretofore described, in varying proportions, and represents by far the great majority of cases found in leper asylums. There may be only a few nodules, with marked nervous changes, or there

may be a large number of nodules with only slight nerve changes. Almost any proportion of these two types may be present.

The only really satisfactory and conclusive diagnosis consists in finding the typical bundles of acid-fast bacilli characteristic of leprosy. As already noted, these can usually be found earlier in a scraping taken from an area from the septum of the nose at the junction of the cartilaginous and osseous portion than anywhere else. The diagnosis is further confirmed by the presence of anesthetic areas, macules characteristic of leprosy, tubercles, loss of eyebrows, and thickening of the ears and nose. Manson[3] mentions that "in doubtful cases further assistance may sometimes be got from the fact that leprous spots rarely perspire. A hypodermic injection of pilocarpin is of use in bringing out this point." Thickening of the nerves in association with some of the other symptoms mentioned is a diagnostic sign of great importance. Likewise, a perforating ulcer of the foot, with other leper symptoms, is also of much value. It is a fact worthy of mention that natives who reside in countries in which leprosy prevails, although they have no medical training, are often unusually proficient in the diagnosis of even incipient cases of the disease.

Until very recently the prognosis of leprosy was most discouraging. Occasionally a case seemed to recover spontaneously or was attributed to some treatment. In more recent years the treatment used in the Philippines has resulted in the cure of at least 10 per cent. of the cases in which it was faithfully carried out, and under similar conditions the clinical disappearance of the disease in at least 25 per cent. of the cases seems a reasonable figure. *It may be stated that the faithful use of treatment as recommended will arrest the progress of the disease in almost every case.*

TREATMENT.

A review of the literature shows that practically the entire pharmacopeia has been called into requisition in an attempt to find a cure for the disease. A trial of these various remedies shows that none of them appear to be of much value except chaulmoogra oil. Of all the remedies used, chaul-



Fig. 1.—Case of leprosy in a child showing infiltration, especially in ears, lips, and hands. Leprous nodules in the left arm. Example of tubercular nodular or hypertrophic leprosy.

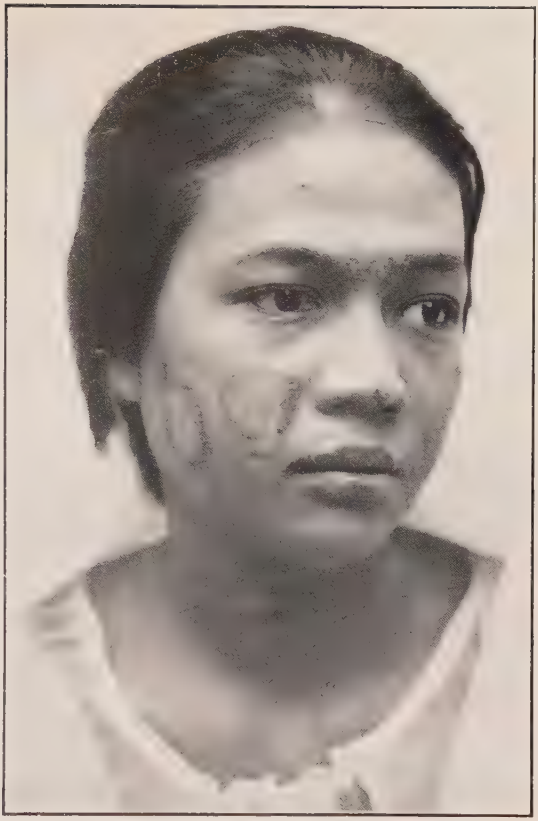


Fig. 2.—Typical macular leprosy.



Fig. 3.—Typical "main en griff" in leprosy.

moogra oil alone has stood the test of time. Owing to the nauseating effects of the oil, when given by mouth, it was never used extensively. A preparation of the oil in which the emetic principle had been removed was tried, but this apparently had no influence on leprosy. Emulsions of different kinds were prepared. Capsules were coated with various substances so as to permit them to pass through the stomach unaltered, but nausea usually followed all of these efforts. It was seldom possible to find a patient who was able to take the prescribed doses by mouth for a period longer than three months. The few who were able to continue usually showed great improvement, and occasionally cures were reported. Enemas of chaulmoogra oil were tried, but these had no apparent influence on the disease. Chaulmoogra oil had been given hypodermically, but it usually failed to be absorbed. To overcome this difficulty camphorated oil was added to the chaulmoogra resorcin prescription of Unna, and this made the administration of chaulmoogra oil practicable. The prescription is as follows:

Chaulmoogra oil,
Camphorated oil .....āā mils 60 (2 f℥).
Resorcin ..... grams 4 (61.7 gr.).

Mix and dissolve with the aid of heat on a water bath and then filter.

The injections are usually made at weekly intervals in ascending doses. The initial dose is 1 mil (16 *m.*), and this is increased to the point of tolerance. Much difference exists among the cases as to the amount of the mixture which they are able to take. In some cases a few mils produce marked reactions in the lesions, accompanied by fever and cardiac distress. Sometimes it is better to reduce the amount of the dose and inject at more frequent intervals. The object sought is so to regulate the dose as to prevent reactions of too violent a character. Quicker results are also apparently obtained when it is possible to inject the mixture into large leprous deposits or to divide the dose by injecting it into a number of smaller infiltrations. Two per cent. hot sodium bicarbonate tub baths are prescribed every other day. Those who take prolonged baths regularly seem to improve more rapidly than those who do not. This treatment should be given over a

period of at least two years, and no case of leprosy should be regarded as cured until it has been continuously negative, both microscopically and clinically, for a period of two years. Recently Vahram, of Paris,[4] has recommended the intravenous injections of 1 mil (16 *m.*) doses of a very finely subdivided chaulmoogra oil, which he prepared by a special process. There are not yet sufficient data available to pass judgment on this form of treatment.

Prevention. While the exact mode of transmitting leprosy is not known, two or three facts stand out so prominently that the course in attempting to prevent its spread and control seems to be entirely clear. It may be asserted without successful contradiction that no cases of leprosy occur in any community in which there has not been a previous human case. Unlike tuberculosis, public opinion, with the exception of a very small minority, will support isolation and segregation measures. As these offer the only hope at present for controlling or eradicating the disease, it is believed to be the duty of medical men rigidly to advocate this policy, and to take all reasonable steps to bring it about. All cases of leprosy, regardless of type, in which it is possible to demonstrate the leprosy bacillus, should be segregated in a leper colony. Such lazaretto should be made comfortable and offer every reasonable facility for the study and treatment of the disease. Those who remain negative, both clinically and microscopically, for a continuous period of two years should be released with the understanding that they will report at monthly or quarterly intervals to their respective health officers for examination. The conclusion is submitted that whatever may be the views of well-informed persons with regard to the communicability of leprosy, and however widely medical men may differ upon this question, yet the incontrovertible fact remains that every leper is a source of danger to others, and at least one center of infection. So long as the exact mode of transmission is not known, it is apparent that an effort to control the disease without eliminating the leper as a center of infection will be doomed to failure. Prophylactic medicine should not be permitted to be turned by a few sentimentalists from its march to a goal which offers the prospect of the eradication of this plague from the earth, and the saving of many



Fig. 4.—Case of macular and nodular leprosy with marked infiltration of the face, before treatment with chaulmoogra oil mixture.



Fig. 5.—Same case as above, after hypodermic treatment with a chaulmoogra oil mixture.

innocent victims annually from contracting this most loathsome disease.

MALARIAL FEVER.

Of the numerous synonyms for the malarial fevers the following are current: Ague, Chills and Fever, Marsh Fever, Remittent Fever, Intermittent Fever, Chagres Fever, Paludism, Paludismo (Spanish), Palustre (French), Coast Fever, and Climatic Fever.

Malaria is a term used to designate a group of specific fevers caused by protozoan parasites belonging to the class Sporozoa. The definitive host is the mosquito, and the intermediate host is man, and possibly other vertebrates. There are three distinct types of malarial fever. The first is caused by the *Plasmodium malariæ,* the second by the *Plasmodium vivax,* and the third by the *Laverania malariæ.*

Malaria is a disease associated with the remotest antiquity. Hippocrates recognized the existence of periodic fevers, and divided them into the quotidian, tertian, subtertian and quartan types. Galen, Celsus, and other Roman writers, also give accurate descriptions of these fevers. Until about the middle of the seventeenth century there was very little advance in the knowledge of malaria. The introduction of cinchona, in 1640, enabled Morton and Tort to separate malarial fevers from other febrile diseases, and also to show that some continued and remittent fevers belong to the same group as the intermittent fevers. Another important advance was the description of the characteristic pigmentation of the viscera in malaria, in 1847, by Meckel. Virchow confirmed this observation. In 1854 Planer noted pigmented cells in fresh blood taken from the finger of malarial patients, but did not comprehend their true nature. The parasitic nature of malaria, which had been suspected for many years by Italian observers, was established definitely by Laveran, in 1880, when he described the eruption of the long mobile filaments from the pigmented cells described by Meckel and Planer. Laveran's observations were soon confirmed and extended by Marchiafava, Celli, Golgi, Bignami and Bastianelli. The next and most important advance was made by Ross, in 1895, when he found malarial parasites in the stomach of the mos-

quito. The importance of the discovery that malarial fever is conveyed by mosquitoes was probably one of the most far-reaching that has ever been made in medicine. The control of malaria has now been definitely placed within the grasp of the sanitarian, and it may be safely predicted that it is possible to save millions in lives, sickness and treasure.

As intimated above, malarial fevers are caused by the parasites *Plasmodium malariæ*, *Plasmodium vivax* and *Laverania malariæ*. These parasites may be spread from one human being to another through the intermediate host, the mosquito, in whose bodies they undergo development. Infected mosquitoes are capable of conveying malarial fever to healthy persons in non-malarial climates. This experiment was actually performed on Sir Patrick Manson's son,[5] when a mosquito that had bitten a malarial subject in Rome was permitted to bite him later in London, whereupon he developed a typical attack of the disease.

The following description may be regarded as giving the generic features common to the three different classes of parasites. These may, again, be divided into three phases.

The malarial parasite, like all true parasites, must adapt itself not only for life inside its host, but also that its continuance as a species may be assured during the passage from one host to another. In man, it exhibits two distinct phases, an intracorporeal stage and an extracorporeal stage. Clinical observation and analogy indicate that there is yet another phase which may be described as a latent phase, whose character as yet can only be conjectured.

Each species of the malarial parasite has its special or more or less definite intracorporeal life span or cycle of twenty-four hours, forty-eight hours or seventy-two hours. Upon examination of malarial blood toward the close of a cycle, or several hours before the occurrence of a paroxysm of the characteristic fever, the parasite may be recognized as a pale disc of protoplasm occupying a larger or smaller area within certain erythrocytes. Throughout this pale body there are numerous intensely black or reddish black particles. This substance has been referred to by some authors as melanin, and by others as hemozoin. The groups of black particles concentrate into one or two larger and more or less central

blocks, around which the pale protoplasm of the parasite arranges itself in minute segments, which finally become well-defined spherules. The blood corpuscle is then destroyed, and the spherules, none of which contain the black particles, fall apart, and, with the black particles, become free in the blood-stream. The leucocytes soon absorb the black particles and many of the spherules. A certain number of the latter escape the leucocytes and attach themselves to other erythrocytes, which they enter. In the interior of these newly infected red corpuscles the young parasites exhibit active ameboid movement, shooting out and retracting long pseudopodia. They grow at the expense of the hemoglobin, so that the erythrocytes gradually become pale through the loss of the hemoglobin. As the parasites grow larger the ameboid movement gradually ceases.

It is well recognized that with the subsidence of the acute clinical symptoms, the malarial parasite may disappear from the general circulation. This is not always necessarily the result of the administration of quinin. It may happen quite independently of this. When it is not attributable to the effect of quinin, the disappearance is, as a rule, only temporary. Usually after an interval of some weeks or months the parasite reappears in the general circulation, and there is a renewal of the clinical symptoms. The location of the parasite during the latent period is unknown, although many believe it to be in the spleen, while others suspect the marrow of the long bones.

If the micro-organism propagates so actively in the human body that it has no opportunity of continuing its species, by passing from one host to another, we are forced to conclude that some provision exists in the economy that enables the parasite to leave and enter successive hosts. It is interesting, then, to study the manner in which the parasite leaves the body, what life it leads outside of the human body, and how it re-enters the human body.

The *flagellated body* is found in all forms of malaria. It is an octopus-like structure, with long, actively moving arms. Although it is composed of the same materials (that is, transparent protoplasm and dark granules), yet it differs in many respects from the ordinary forms of the parasite, especially in

that it is not intracorpuscular. It floats free in the liquor sanguinis. It has usually from one to six long whip-like arms, designated as flagella, or, more correctly, microgametes. Their movements are so vigorous that they frequently double up or otherwise disturb temporarily those corpuscles with which they chance to come in contact. Occasionally one or more of these flagella break away and swim free in the blood. If kept warm they may remain active for several hours. Special attention should be drawn to the fact that these flagellated bodies are never seen in the freshly prepared specimen. They come into view only after a slide has been mounted for some time, say from ten to thirty minutes, or even longer. Upon the examination of malarial blood, after it has been mounted as a wet preparation, it is not unusual to observe the flagellated body. Observation has shown that the flagellated bodies are developed during a particular phase of the intracorpuscular parasite.

The bodies known as *crescentic forms* are not present in the blood at the commencement of the malarial infection, or at the beginning of a recrudescence of a latent infection. They appear in the blood after a week or ten days of acute clinical symptoms. At first they are few and difficult to find, but gradually they become numerous, and persist for days after the disappearance of other forms of the parasite. They are not affected by quinin. They may disappear from the blood in a week, or persist for six weeks or longer. At times they may be so numerous that several may be seen in every field of the microscope. Again, they are so scanty that many preparations are necessary before one can be found, and at times it is impossible to find them. The crescent bodies and the large intracorpuscular forms are sexual in their functions. The protoplasm in the crescent differs in the arrangements of its pigment, and the characters of the nucleus are revealed by staining, which are distinctive of the male and the female crescents, respectively. The protoplasm of the male parasite stains more deeply and its nucleus is larger than that of the female parasite.

If a number of crescent bodies are kept under observation on the microscope slide, a certain number of them will be seen slowly, or at times rapidly, to undergo a change of

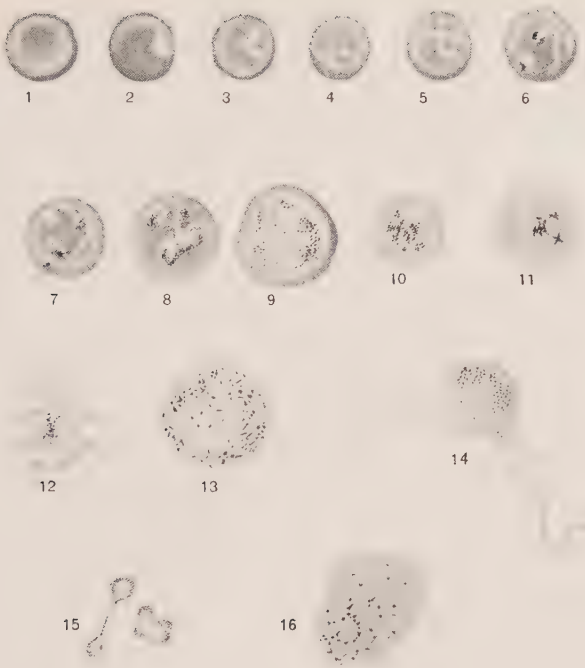


Fig. 6.—The tertian parasite.

1. *Normal erythrocyte.*
2, 3, 4, 5. *Intracellular hyaline forms.*
6, 7. *Young pigmented intracellular forms.* In 6 two distinct parasites inhabit the erythrocyte, the larger one being actively ameboid, as evidenced by the long tentacular process trailing from the main body of the organism. This ameboid tendency is still better illustrated in 7, by the ribbon-like design formed by the parasite. Note the delicacy of the pigment granules, and their tendency toward peripheral arrangement in 6, 7, and 8.
8. *Later developmental stage of 7.* In 7, 8, and 9 enlargement and pallor of the infected erythrocyte become conspicuous.
9. *Mature intracellular pigmented parasite.*
10, 11, 12. *Segmenting forms.* In 10 is shown the early stage of sporulation—the development of radial striations and peripheral indentations coincidentally with the swarming of the pigment toward the center of the parasite. The completion of this process is illustrated by 11 and 12.
13. *Large swollen extracellular form.* Note the coarse fused blocks of pigment. (Compare size with that of normal erythrocyte, 1.)
14. *Flagellate form.*
15. *Shrunken and fragmenting extracellular forms.*
16. *Vacuolation of an extracellular form.*

NOTE.—The original water-color drawings were made from fresh blood specimens, a Leitz $^1/_{12}$-inch oil-immersion objective and 4 ocular, with a Zeiss camera-lucida, being used

(E. F. FABER, *fec.*)



Fig. 7.—The quartan parasite.

1. *Normal erythrocyte.*
2. *Intracellular hyaline form.*
3. *Young pigmented intracellular form.* Note the coarseness, dark color, and scantiness of the pigment granules.
4, 5, 6, 7. *Later developmental stages of 3.* Note the peripheral distribution of the pigment in all the parasites from 3 to 8. (Compare size of the erythrocytes in 5, 6, and 7 with 7, 8, and 9, Fig. 6.)
8. *Mature intracellular form.* Note that the stroma of the erythrocyte is no longer demonstrable.
9, 10, 11. *Segmenting forms.* In 9 are shown the characteristic radiating lines of pigment. (Compare with 10, 11, and 12, Fig. 6, and with 10, 11, and 12, Fig. 8.)
12. *Large swollen extracellular form.* (Compare with 13, Fig. 6.)
13. *Flagellate form.* (Compare with 14, Fig. 6.)
14. *Vacuolation of an extracellular form.*

(E. F. Faber, *fec.*)



Fig. 8.—The estivo-autumnal parasite.

1. *Normal erythrocyte.*
2, 3. *Young hyaline ring-forms.*
4, 5, 6. *Intracellular hyaline forms.* In 4 the parasite appears as an irregularly shaped disc with a thinned-out central area. In 5 and 6 its ameboid properties are obvious.
7. *Young pigmented intracellular form.* Note the extreme delicacy and small number of the pigment granules. (Compare with 6, Fig. 6, and with 3, Fig. 7.)
8, 9. *Later developmental stages of 7.*
10, 11, 12. *Segmenting forms.*
13, 14. *Crescentic forms at early stages of their development.*
15, 16, 17, 18, 19. *Crescentic forms.* In 15 and 19 a distinct "bib" of the erythrocyte is visible. Vacuolation of a crescent is shown in 18, and polar arrangement of the pigment in 17.
20. *Oral form.*
21, 22. *Spherical forms.*
23. *Flagellate form*
24. *Vacuolation and deformity of a spherical form.*
25. *Vacuolated leucocyte apparently enclosing a dwarfed and shrunken crescent.*
26. *Remains of a shrunken spherical form.*

(E. F. Faber, *fec.*)

shape, gradually being converted into flattened crescents, then into oval bodies, and then into spheres, while the remains of the enclosing blood corpuscle fall to pieces or dissolve.

That some relationship existed between the mosquito and malaria has long been entertained, not only by medical men, but also by inhabitants of malarious countries. In the Roman Campagna, for instance, the peasants have believed for centuries that the disease is produced by the bite of the mosquito. Koch pointed out that the natives of East Africa, who inhabit the highlands, declare that when they visit the unhealthy lowlands and are bitten by an insect which is presumably the mosquito, a fever results.

In 1894, and again in 1896, Manson[6] formulated a definite hypothesis on the subject. He expressed the opinion at that time that the parasite, in order to maintain its existence as a species, must pass from host to host. In other words, it must at times have an extracorporeal life. He concluded that the function of the flagellum lay outside of the human body, and that the flagellated body was the first phase of the extracorporeal life of the malarial parasite. As the parasite, while in the circulation, is always within a blood corpuscle and is incapable of leaving the body by its own efforts, and, so far as is known, is not excreted, Manson concluded that it must be removed from the circulation by some blood-sucking insect. Manson based his arguments on what he had shown to be the case with regard to the *Filaria bancrofti,* and reasoned that, in all probability, a particular type of mosquito was responsible. He suggested the investigation of this hypothesis to Ross, who in 1897 was able to offer definite proof of the correctness of the hypothesis.[7] A gap in Ross's observation was filled in by MacCallum, who showed by observations of a malaria-like parasite in birds that the function of the filament after it breaks away from the parent sphere or flagellated body, is to impregnate the granular crescent-derived spheres and then to be transformed into sharp-pointed traveling vermicules.

The work of Ross was soon confirmed and elaborated by Daniels and by Koch, as well as by a number of Italian observers. Grassi showed that several species belonging to the genus *Anopheles,* and in Italy the *Anopheles maculi-*

pennis, are the special mosquito hosts of the malarial parasites of man. On behalf of the Colonial Office and the London School of Tropical Medicine, with the assistance of Dr. Sambon and Dr. Low, Dr. Manson instituted two experiments which resulted in silencing all the objections which had appeared against the theory.[8] Dr. Sambon and Dr. Low lived for the three malarial months of 1900 in Ostia in the malarial part of the Roman Campagna, in a hut from which mosquitoes were excluded by wire screens. They moved about freely in the neighborhood during the day, exposing themselves in all weathers, drank the water of the place, and often did hard manual work. The only precaution they took was to retire between sunset and sunrise to their mosquito-protected house. They took no quinin. Their neighbors, the Italian peasants, were attacked by malaria, but the dwellers in the mosquito-proof house enjoyed immunity from the disease. While this experiment was in progress, mosquitoes fed in Rome on patients suffering from tertian malaria were forwarded in suitable cages to the London School of Tropical Medicine, and on their arrival were permitted to bite the son of Dr. Manson and Mr. George Warren. Shortly afterward both of these gentlemen, neither of whom had been abroad or otherwise exposed to malarial influence, developed characteristic malarial fever, and malarial parasites were found in abundance in their blood, both at the time and at the recurrence of the fever from which they subsequently suffered. The mosquito theory has, therefore, now passed from the region of conjecture to that of fact.

By a long series of experiments it has been ascertained that only the mosquitoes which belong to the *Anophelinæ* group are capable of transmitting an infective parasite to the human host. When the two types of hyaline and granular crescent bodies—that is, the male and female—are injested with blood of man by *Anopheles* mosquitoes, they soon emit filaments or microgametes, which break away and bore into the granular spheres. Soon after this impregnation takes place an elongated oval results, which gradually assumes a vermicular form. The hemozoön accumulates on the posterior end, while the anterior end becomes pointed and hyaline. On the completion of these changes this little body begins to

move about, first slowly and then rapidly. This traveling vermicule is technically known as an oökinete, and it soon passes into a white or a red blood corpuscle. Soon afterward the oökinete penetrates the wall of the mosquito's stomach, where it may be found thirty-six hours after it has been injested by the mosquito. During the next few days the parasite increases rapidly, acquires a well-defined capsule, and soon protrudes on the surface of the insect's stomach. During this period important changes take place in the interior of the parasite, and the term oöcyst is now applied. The nucleus and protoplasm divide into a number of spherical daughter cells, around which, attached by one end, like the spines on a porcupine, a vast number of minute, slender, spindle-shaped, nucleated bodies are ultimately formed. At a later stage the spherules disappear and leave the spindles, the so-called sporozoïtes, loose in the capsule, which is now packed to the bursting point. In about a week—and the period depends much on the atmospheric temperature—the capsule ruptures and collapses, and discharges its contents into the body cavity of the mosquito.

From the body cavity of the mosquito the sporozoïtes pass by the way of the blood to the salivary gland of the mosquito, which lies on each side of the forepart of the thorax of the insect. These glands communicate with the base of the mosquito's proboscis by means of a long duct. When the mosquito bites man these sporozoïtes are injected during the act of biting, and eight or ten days later the malarial parasite may be found in the circulating blood of the infected man.

So far as is known at present the malarial fevers can be conveyed to man only by the means of mosquitoes. However, there are instances which arise which make it difficult to understand how mosquitoes and man alone can be responsible. It has been frequently reported that in certain areas of the world which are entirely uninhabited by man, a malarial infection may be contracted by a visit to such areas. It has also been pointed out that malaria frequently occurs where public works are undertaken which are attended with disturbances of the soil, as in railroad building, canal work, harbor works, etc. Faulty observation probably explains many of these instances. Mosquitoes which have had access

to man are often present without actually being observed. Other instances are usually explained on the basis that there is a possibility of another host than man for the malarial parasite. Parasites closely resembling human malarial organisms have been found in bats, and also in monkeys. Another explanation submitted is that malarial parasites may be transmitted from one generation of mosquitoes to another through the means of their eggs. There is the support of analogy for this hypothesis. *Babesia bovis,* which gives rise to Texas fever in cattle, and *Babesia canis,* which causes malignant jaundice in dogs, are both said to be transmitted through the means of eggs. In general, however, it may be stated that most observers are of the opinion that under natural conditions malaria can be acquired by man only through the bite of a mosquito, and that a mosquito can obtain its infection only by biting an infected human being.

For the microscopic examination of the blood, several preparations, as a rule, should be made, the blood being obtained by gentle compression of the finger-tip or the lobe of the ear, and a puncture then made with a needle that is triangular on cross section. Antiseptic precautions, of course, should be observed. A droplet of blood should be collected on a cover-glass, care being taken that the glass does not come in contact with the skin. The cover-glass should then be dropped upon the glass slide. Pressure to cause the blood to spread out should not be used. If the glasses have been well cleaned, the blood will at once run out in a fine film, showing the flat surfaces of the corpuscles, lying edge to edge. After waiting a few moments for the blood to spread out it is well to ring the preparation with vaseline. This will stop all movement, evaporation and overcompression of the corpuscles, and will greatly facilitate examination. Accuracy can be acquired only by practice, and those examining blood should learn the art from one who has had considerable experience. Further details of the microscopic technic and staining methods can be obtained from various laboratory manuals.

The different species of malarial parasites have been classified in accordance with (*a*) the duration of their respective life circles inside the human body, (*b*) their morphologic charac-

ters, (*c*) the clinical phenomena to which they give rise, and (*d*) the results of inoculation experiments.

In the first place, malaria may be divided into the benign and the malignant type. Morphologically, the benign parasites do not form crescent bodies, and the most important malignant parasites do form crescents. The gamete of the benign parasite is a sphere or a disc, and the gamete of the malignant parasite is a crescent. Clinically the benign parasite does not give rise to pernicious attacks, while the malignant parasite usually does. The benign parasites are of two kinds: (*a*) the quartan parasite, having a cycle of seventy-two hours, causing a recurring fever every three days, so-called quartan fever; (*b*) the tertian parasite, which has a cycle of forty-eight hours, and causes recurring fever every two days, so-called tertian fever. The malignant parasite has three forms, and, perhaps, more. The pigmented parasite causes so-called subtertian fever of forty-eight or approximately forty-eight hours' cycle. Another pigmented parasite, often called the pigmented quotidian, has a twenty-four hours' cycle, and is referred to by some authors as double tertian.

Formerly classification was based entirely upon clinical phenomena, and referred to as quotidian, tertian and quartan intermittent fever or ague, and remittent fever. But since it has been found that remittent fevers are produced by either quartan, tertian, subtertian, or by quotidian parasites, it is believed that the classification given above is more desirable. It is quite likely that further study of the disease will bring about a more satisfactory classification than exists at the present time.

Typical attacks of intermittent malarial fever, and most of them are typical in persons infected for the first time, consist of a series of phenomena which recur at definite intervals. Each attack consists of a chill, a period of fever, and a period of sweating; and these are succeeded by an interval of apyrexia. There is much variation in the duration and intensity of the different stages. As a rule, there is a certain proportionate relationship. Often the more pronounced the chill, the higher the fever, and the more profuse the sweating. The expression "ague" is applied only to intermittent fever with which there is a pronounced chill.

The geographical distribution of the malarial fevers is widespread. The tertian is probably the most common form, and occurs in temperate and tropical latitudes alike. The pernicious forms of the fever are rarely found outside of the tropical belt. Generally speaking, malaria is most virulent in the region of the Equator, and decreases numerically and in virulence as the distance from the Equator increases. The malarious area may be said to lie between 63 degrees north latitude and 35 degrees south latitude. Many countries that suffered severely from the disease in the past now are apparently becoming free of malaria. For instance, except in limited areas, the disease is not known to occur at present in England, Holland, France and Germany. In former times malaria prevailed in all of these countries to a very considerable extent. In the United States the northern line of the disease is gradually receding. Some years ago malaria was very common in Wisconsin, Minnesota, Iowa and other northern States. At present there is practically no malaria in those regions. It is rather rare to find a case north of Mason and Dixon's line. On the other hand, malaria is appearing in many regions in which it has not occurred heretofore. This is due to improved means of travel, through persons in the infective stage taking up residence in places in which *Anopheles* mosquitoes occur. Instances of this kind were very common in the Philippines, and it was often noted that when troops that had been stationed in malarious districts were transferred to other villages, provinces or islands, in which malaria had not formerly prevailed, the disease soon appeared among the inhabitants. Sugar plantations, public works, and other places where malaria prevailed often recruited laborers from islands that were free from malaria, and later when these laborers returned to the islands they caused outbreaks of malaria. In accordance with a recent survey made by Trask[9] based on reports received from State and city health officials, the distribution of malaria in the United States was included in the following areas: "The large endemic area covers the whole southeastern portion of the United States, having for its southern boundary the Gulf of Mexico; for its western boundary, a line drawn from Eagle Pass, on the Rio Grande, to Leavenworth, Kan.; for its eastern boundary the Atlantic

seaboard; its northern boundary, a line drawn from Leavenworth, Kan., eastward some distance north of the Ohio River and extending to the Atlantic on a line with the northern boundary of Maryland. Of the two smaller endemic areas, one includes a section of the northern part of New Jersey, southeastern New York, Connecticut, Rhode Island, and part of the State of Massachusetts. The third recognized endemic area is in California, and includes the Sacramento and San Joaquin Valleys, which occupy a large portion of the central part of the State. It is probable that the New England endemic area actually extends southward to the large southern area of which it is in reality a part." These surveys showed that there was one large and two smaller areas.

The mosquito malaria theory, which is now thoroughly established, furnishes the key to the etiologic problem. Whatever favors the presence and increase of malaria-bearing mosquitoes also tends to increase the incidence of malaria, provided that there are human beings in the vicinity whose blood harbors the malarial parasites. The disease is caused by the *Plasmodium malariæ* of Laveran, by the *Plasmodium vivax,* and the *Laverania malariæ*. These parasites are always found in the blood or organs of the person suffering from the disease, and can be injected into healthy persons, in which they cause typical fevers, which occur in different stages and correspond to the life cycle of the particular parasite which has been injected. There are three factors necessary for the production of malarial fever: the blood parasite, the mosquito and man.

Malarial fever is found in the northern hemisphere from the Arctic Circle to the Equator. In the southern hemisphere its distribution is perhaps equally great, although not yet so well proved. The disease is not uniformly distributed throughout this vast area. It occurs in limited endemic foci, and, as pointed out above, the disease tends to become more virulent as the Equator is approached.

The relationship between temperature and mosquitoes is very definite. Practically no cases of malaria occur after sufficient frosts have taken place effectually to kill all mosquitoes. New cases do not occur again until weather conditions favor the breeding of mosquitoes. There are certain

countries, although in the tropical belt, in which malaria does not occur. For instance, the Fiji Islands and Barbados are typical examples. A careful mosquito survey made in both these countries shows a complete absence of the *Anopheles* mosquito. Rainfall and conditions of moisture apparently have no influence on the disease, except in so far as they promote the breeding of mosquitoes. Likewise, winds and atmospheric diffusion are only important in the etiology of the disease, in so far as they may be the means of either preventing the flight of mosquitoes, or assisting in the distribution of the infected insects.

In times past it was often thought that certain trees, as, for instance, eucalyptus trees, were protection against malaria. It is more than likely that this apparent protection may have been due to the drying influences on the soil which eucalyptus trees possess owing to the large amount of water which they withdraw. The odor of the trees may also have deterred mosquitoes from coming near. It is also possible that they may have given protection by forming wind-breaks, and thus interfered with the flight of mosquitoes from swamps or other insect-breeding grounds.

The time of the day in relation to infection also conforms very closely to the habits of the mosquito. *Anopheles* usually fly only between sunset and sunrise, and persons can ordinarily go into malarious districts during the daylight hours without any great danger of contracting the infection.

It has been explained by Koch and others that the apparent immunity which exists among Negroes, Melanesians and other dark-skinned races living in highly malarious countries, is in all probability due to the resistance they have acquired during the constant attacks they suffered in childhood. It has been observed that the percentage of infected children gradually becomes smaller as their age increases.

When a malarial infection occurs, the body is invaded by protozoal parasites which grow and increase at the expense of the red cells of the blood, and upon these essential premises the pathologic changes incident to the disease are based. Two toxins have been isolated—pyrogenetic toxin and hemolysin. Erythrocytes are found in all the circulatory organs, and are generally within the blood-vessels. In the spleen and in the

bone-marrow, however, they come into intimate relationship with the parenchyma. It would seem that, as the parasites are distributed by the erythrocytes, they should be found in all of the organs. But this condition does not usually follow, especially in pernicious infections. When it does not occur the parasites probably damage the red blood-corpuscles, and through the toxins set free, the endothelium of the vessels is damaged, especially that of the capillaries, and blocking of the blood-stream occurs, which prevents even distribution. Apparently the damage done to the erythrocytes by the quartan parasites is not severe enough to cause their stagnation in the capillaries. Therefore, they are more evenly distributed than the other types. Tertian parasites cause the erythrocytes to undergo swelling, degeneration and decolorization. The subtertian parasites also seriously affect the red cells, and make them smaller and darker. Subtertian parasites are seldom found in the peripheral circulation in the sporulating stage. The pigment which remains after the red corpuscle is destroyed may be found in the peripheral blood in a free state, and in the mononuclear leucocytes. Hemolysin also escapes from the sporulating parasite, damages the erythrocyte and causes the appearance of another pigment, yellowish in color, called hemosiderin. This is deposited in the parenchyma cells of the organs, especially in the liver. The damage done to the parenchyma is but slowly repaired, and considerable time elapses before permanent recovery takes place. In the case of the subtertian parasite, serious local damage may be done to the brain, the intestine, the pancreas, or other organs in which the parasites may mass themselves within the capillaries.

There are two main distinctions to be made in the pathology of malaria. First, that of acute malaria; and, second, that of chronic malaria. In acute malaria the effects are produced by each of the three parasites, of which the subtertian is liable seriously to damage important organs. Chronic malaria is encountered in a condition called malarial cachexia, which manifests itself in the form of acute cachexia, chronic cachexia, and cachexia with amyloidosis.

Rosenau and his collaborators proved the poisonous properties of the pyretogenous toxin. It is not definitely known

whether this poison has deleterious influences upon the tissues of the organs.

Iron is excreted through the urine in increased quantities. This increase does not appear, however, until several days after the actual attack is over. The chlorides are diminished. The phosphates are increased. The sulphates are higher than normal, but during the actual attack they are lower than normal.

Iron is also excreted in considerable quantities through the feces. The sweat of malarial patients has a peculiar odor and is toxic to rabbits. Hemozoin is the black pigment formed from the hemoglobin by the malarial parasites while they are in the red blood-cells. This pigment is later distributed to the different organs, and accumulates, as a rule, in large quantities in the liver and spleen. Hemosiderin is the yellow pigment usually found in the parenchyma cells of the liver, spleen, kidney, bone-marrow, and endothelium of capillaries.

The malarial parasites in the blood produce changes in the red cells by their own action and through their toxins. They are the most important feature in the pathology of the blood. Ross[10] estimates that the average person weighing 150 pounds (68 Kg.) possesses 25,000,000,000,000 erythrocytes. In a severe infection he estimates that 12 per cent., or 3,000,000,000,000 corpuscles, are affected. It also has been quite definitely demonstrated that large numbers of parasites may exist in the body and go through their life cycle in the spleen without causing noticeable clinical symptoms. This type of disease is usually referred to as latent malaria.

The most characteristic pathologic signs of malaria at autopsy are slatish- or bluish- black pigmentation, which affects mostly the spleen, liver, brain, and sometimes the intestinal mucosa. The spleen is always enlarged and usually quite soft. The liver is enlarged and congested. Pigmentation is most pronounced in those cases that have died after protracted infection. In the brain sometimes the capillaries are found to be actually plugged with masses of erythrocytes which contain parasites, together with phagocytic cells. Occasionally free parasites also are found in the blood. A similar condition is sometimes found in the capillaries of the

mucosa of the intestine. Some authors report total necrosis in the liver, and there are nearly always parasites in the capillaries. The spleen is practically always heavily infested with parasites, which may readily be demonstrated in stained microscopic sections. The *Plasmodium vivax* seldom causes death, but, as a rule, there are other lesions due to intercurrent disease.

In undertaking a description of the *symptomatology* of malarial fever it should be kept in mind that malaria is due to three distinct parasites, namely, *Plasmodium malariæ*, *Plasmodium vivax* and *Laverania malariæ*. These parasites give rise to three clinical entities, namely, quartan malarial fever, tertian malarial fever and subtertian malarial fever, but there are a number of subdivisions of these different varieties. The quartan and tertian parasites have their whole life history in the circulating blood, and while the tertian sporulating forms may be found in the internal organs, spleen or other internal organs, yet they do not accumulate or produce special effects in those organs. The subtertian parasites sporulate almost entirely in the internal organs, and it is this sporulation in an organ which gives rise to the special clinical features which have been described as the pernicious types of malarial fever. The particular variety of fever will depend upon whether the parasite is localized in the cerebrospinal nervous system, in the gastro-intestinal mucosa, or in the pancreas, the heart, the lungs, or the liver.

The Quartan Fevers. Quartan fevers depend upon the *Plasmodium malariæ* which has been introduced into the blood by the *Anophcles* mosquito, and the clinical course will depend upon whether the parasites are approximately the same age or whether they have been introduced into the body on different days. If malarial organisms of about the same age have been introduced into the blood, they will give rise to typical quartan malarial fever, which has an interval of seventy-two hours between the paroxysms. This corresponds to the period required by the merozoïte to reach the fully developed schizont. This type of fever is known as simple quartan. If the parasites have been introduced on different days, and also are perhaps of different ages, the patient will develop fever on two successive days and be free on the third

day. Such a fever will be called a double quartan. From the foregoing it is evident that many combinations are possible, depending upon the intervals of introduction and the age of the parasites.

Simple Quartan Fever. The incubation period of fever due to this parasite has not been definitely determined. Celli, by experiment, came to the conclusion that perhaps the incubation period was several months or more. By experimental inoculation of blood, other Italian workers estimated that the maximum incubation period was eighteen days, and the minimum eleven days.

The Fever Stage. As a rule, several hours before the rise of temperature occurs, the patient may complain of dizziness, weakness, malaise, headache, and sometimes nausea and vomiting. If the blood should be examined during this period the parasites would be found to be schizonts. At the expiration of several hours the definite attack begins, which may be divided into three stages: first, the chilly; second, the fever; and, third, the sweating.

The Cold Stage. A chilly sensation is felt in the legs, arms or back. This increases until actual shivering sets in. The rigors are well marked and characteristic, and it is not infrequent that the patient will actually shake the bed. The teeth chatter, the lips become blue, the arms and legs cold, and goose-skin may be present. The cold stage is by far the most uncomfortable. The internal temperature rises rapidly, and congestion of the internal organs usually takes place. The temperature may vary from 100° to 105° F. (37.7° to 40.5° C.). The chilly stage usually does not last more than thirty minutes, although it may be much shorter or even longer.

The Hot Stage. The shivering gradually ceases and the patient begins to feel warmer and more comfortable, although there are waves of hot and cold sensations that pass through the body. The patient begins to throw off the bed-clothes. The skin feels hot and dry, and the frequency of the pulse and respirations increases. Vomiting and diarrhea may occur, and a red flushing, especially of the face and neck, frequently may be observed. The temperature remains at its maximum until the latter part of this stage, which usually lasts from three to four hours.

The Sweating Stage. It may be noted that perspiration begins to gather about the forehead and gradually appears all over the body. The patient begins to feel more comfortable. The temperature falls rapidly and the pulse rate declines. Very often the patient falls into a deep sleep, and the symptoms generally disappear.

The Interval. If the patient has gone to sleep, he usually awakens feeling quite refreshed, although weak, and goes about his ordinary work during the entire two days' interval. Examination of the blood at this stage shows that there may be leucopenia, and also developing parasites. At the end of the seventy-two hours the fever again rises, and there is a repetition of practically all the symptoms described above. There are, of course, many irregularities in the appearance of the symptoms, but the description given corresponds more or less to the typical case.

Quartan fever is usually regarded as having a great tendency to relapse and to reappear even at intervals of years, if it has not been adequately treated. Pernicious symptoms seldom appear. If the disease remains untreated the fever gradually disappears, but recurs at times. Complete spontaneous cure is regarded as being rare.

Double Quartan Fever. In this form of malaria there is an attack on two successive days, and an interval of freedom from fever of twenty-four hours. Otherwise, the symptoms are much the same as those described above.

Triple Quartan Fever. This form is due to triple infection, and there are daily paroxysms of the kind described above, the interval lasting only a few hours.

Tertian Fevers; The Simple Tertian. This fever recurs every forty-eight hours, with apyrexial intervals of a day. Prodromata may or may not occur. In some instances they are quite characteristic, and take the form of pain in the head and back, especially in the bones of the limbs, particularly in the joints. These pains are often mistaken for rheumatism. There is a feeling of lassitude and illness. On the day succeeding the foregoing symptoms the patient may feel quite well, and the day following the symptoms may begin to recur. If treatment should take place in this stage, fever may never occur. If untreated, after dizziness, nausea or vomiting, and

some rise in temperature, the patient suddenly becomes very cold and shivers, and the symptoms resemble those described for quartan malarial fever, except that they are not so severe. In less than half an hour the warm stage begins. Soon the patient feels burning hot, the skin is flushed and dry, the eyes are injected, the pulse is quick and often dicrotic, and the pains in the head and back increase. Not infrequently in the white race there is a sallow or slightly yellowish tinge. In the native races this can only be observed in the conjunctiva. The spleen is enlarged and tender. The temperature begins to rise before the chill, and may reach 105° F. (40.5° C.). The whole attack usually lasts from ten to twelve hours, and generally begins in the morning, but may take place any time of day.

Double Tertian Fever. When malarial organisms mature on separate days there may be paroxysms every day. This type of fever is referred to as the double tertian. The symptoms are similar to those before described.

If tertian malaria remains untreated it has a tendency toward spontaneous cure, but relapses may occur from time to time. The anemia of tertian fevers apparently yields more readily to treatment, or even to self-cure than that due to other malarial parasites.

Subtertian Fever. In subtertian fever, although it is a tertian fever, the attacks are due to the *Laverania malariæ*, and are very much more prolonged than those due to the tertian parasites. At least six different types of subtertian fever are described by various authors.

Simple Subtertian Fever. According to Marchiafava and Bignami the incubation period, when transmitted by the mosquito, is from nine to ten days; and longer when acquired by experimental mosquito infection. The chilly stage of the fever may be entirely absent, although there are instances in which it is severe. Very often the attack begins with a warm stage, accompanied by severe pains in the limbs, back and head, with vomiting and diarrhea. The skin is usually flushed and dry, and sometimes somewhat jaundiced. The sweating stage is usually well marked. The spleen, as well as the liver, is tender. The hourly temperature chart is of great diagnostic value. The invasion usually begins with a tempera-

ture of 104° to 105° F. (40° to 40.5° C.), with hourly oscillations of one degree Fahrenheit. Usually the oscillation, which immediately precedes the crisis, is larger, and is often referred to as the pseudocrisis. It is after this precrisis that the temperature reaches its highest point, and then falls very suddenly. In brief, the symptoms largely correspond to an irregular type of tertian fever.

Double Subtertian Fever. This is a daily fever, caused by two invasions of the *laverania* malarial parasite. The symptoms resemble very closely those described before. Other forms of subtertian fever are described as irregular subtertian fever, remittent subtertian fever, in which the temperature during the interval does not reach normal, and also a bilious remittent fever. This derives its name from the fact that it is associated with jaundice, much vomit of bile, and usually a bilious diarrhea. It is due to the fact that there has been great erythrocyte destruction with consequent bile production. Another variety is described under the head of pernicious subtertian fever. The subtertian parasite may pass its life history in the capillaries of some particular organ, and in that event the clinical symptoms are more or less localized. However, there may be pernicious malaria due to large numbers of parasites in the general circulation. This latter type may be divided into the algid pernicious, the diaphoretic pernicious, the hemorrhagic pernicious, and the scarlatinaform pernicious. The type with local symptoms may be divided into the cerebrospinal, the gastro-intestinal, the cardiac and the pulmonary.

The Algid Form. The patient usually is in extreme collapse, and may present many of the symptoms of cholera. The cheeks are sunken, the lips are cyanotic, the nails are blue, the pulse is small and soft, frequently becoming thready and imperceptible, the skin is cold and clammy, and the respiration is labored. This is a very fatal form of pernicious malaria, and usually the patient dies in a few hours.

The Diaphoretic Type. In this fever the sweating of the third stage is profuse. It often happens that not only the bed is saturated, but a pool of water may be found on the floor. The patient soon becomes completely exhausted, and collapse may occur at any time.

The Hemorrhagic Pernicious Type. This type of the disease is rare, and is characterized by hemorrhages of the skin and the mucous membrane of the nose, bronchi, intestines, stomach and generative organs. These hemorrhages occur during the fever stage. This disease rapidly produces serious anemia, ending in coma, delirium, convulsions and death.

The Scarlatinaform Type. This is characterized by a scarlatinaform rash which appears all over the body, with desquamation. If not treated, it usually leads to the typhoid state, in which the patient succumbs.

The Cerebrospinal Type. Very often symptoms of hemiplegia are present. In malarious districts in cases of apparent apoplexy blood examinations should invariably be made for malarial parasites. The fever in this type of the disease may be comatose in character. Usually the patient comes under the observation of the medical man in an unconscious condition, with no paralysis and no alteration in the reflex. The pupils may be markedly contracted and resemble those in opium-poisoning. In cases in which the prognosis is grave, the patient becomes colder and colder until death takes place. Hemorrhages are usually found in the skin and the retina; albumin and casts in the urine. There are many variations of the comatose type. They may take the form of delirium, tetanic or eclamptic convulsions, hemiplegia, aphasia, amaurotic symptoms. Bulbar symptoms may also occur. Sometimes ataxic symptoms are very pronounced. In brief, there may be nervous symptoms which resemble any of the well-known nervous diseases due to pressure in the brain.

Gastro-intestinal Types. Some forms of pernicious malarial fever closely resemble cholera, and may be accompanied by marked vomiting and diarrhea, with abdominal pains, subnormal temperature, and other symptoms characteristic of cholera. An examination of the blood will usually reveal the parasites. The temperature does not remain subnormal for a prolonged period of time. Sometimes the symptoms resemble those of severe dysentery or hemorrhagic pancreatitis, or even those of pneumonia or pleurisy; the latter when there is stagnation of the parasites in the capillaries of the lungs or of the pleura.

Chronic Malaria. Chronic malaria may result from any of the three types of malarial parasites, but, as a rule, chronic malaria is caused by the *Laverania malariæ*. Under this head is also usually included the clinical entity known as malarial cachexia. The symptoms of chronic malaria are repeated attacks of slight fever, which may pass unnoticed. There is enlargement of the spleen and liver, and pigmentation of the skin and mucosa. Frequently there is pigmentation in the tongue, which makes this condition more readily recognizable in the dark races. The pale jaundice condition of the skin in persons who have lived in malarious districts over long periods of time is most characteristic. Briefly, chronic malaria resembles a mild attack of the benign fevers which has been described above.

In malarial cachexia the intervals between the fever are very long, and may be of several weeks' duration. During these intervals it is almost impossible to find the malarial parasite. When the mild febrile attacks occur the parasite can usually be found in the blood. This condition is frequently mistaken for uncinariasis. Chronic malaria and malarial cachexia usually result when there has been insufficient treatment, or when no treatment has been carried out.

Relapses. Malarial relapses in those who have suffered from the disease are frequent. They often occur in persons who may have been free from the disease for months, and even for years. They are specially common among those who have had malaria in the tropics and then go to a cooler climate. Soon after being subjected to a lower temperature, typical attacks of malaria may occur. This is often noticed in the tropics when infected persons, who have been living in malarious lowlands for a long period, go to the mountains where the temperature is low. In this type of attack reinfection in all probability has not taken place. In addition to the relapses which occur upon change of climate, relapses in cases that have not been adequately treated are also frequent. These occur from twenty days up to a number of months after the original attack has occurred.

Reinfection. Persons who are constantly exposed to mosquitoes that have bitten cases of malaria naturally have many opportunities to become reinfected, and there is much evi-

dence that this actually takes place. Previous attacks apparently give no immunity to fresh infections, or at least very little protection, especially if these infections happen to be with parasites of a different type from those from which the original infection took place.

Complications. Complications in malaria are rather common. Typhoid fever, for instance, has been frequently associated with malaria, and has caused great literature on this problem to appear. For instance, during the time of the Spanish-American War, it was contended by one set of physicians that the large sick rate which occurred in Cuba was due to malarial fever with typhoid symptoms, whereas another group of physicians contended that it was an anomalous form of typhoid fever. It is more than likely that in many of these instances there was an infection with typhoid as well as with the malarial parasite. Briefly, malaria and typhoid may exist in the same individual, but it is not believed that this occurs very often. With modern laboratory aids it is not likely that any great difficulty will exist in making the proper distinction. Dysentery, both the bacillary and the amebic, being a common tropical disease, frequently is associated with malarial fever. There is still considerable dispute as to whether there is a true malarial pneumonia, or whether the pneumonia, which is frequently associated with malaria, is due to the regular pneumonia organisms which, perhaps, are able to invade the human host, on account of the lowered state of resistance incident to attacks of malarial fever. Nephritis is commonly associated with tertian and subtertian fevers, and is probably caused by the irritation to the kidney by the malarial toxins.

Sequelæ. The sequelæ of malarial fever are numerous, and may occur in the nervous system, the sense organs, the blood, the liver and the spleen. In the nervous symptoms, insanity, melancholia or other psychoses are not infrequent. Neuritis of malarial origin is very common, and often is associated with distressing symptoms. Neuralgia is a frequent sequela, and may persist for years. It is considered doubtful whether tinnitus aurium, vertigo, deafness, amaurosis and loss of taste are actually due to malaria, or whether they are due to overdoses of quinin. Cirrhosis of the liver, according to many

authors, may follow a prolonged attack of malaria. Characteristic enlargement of the spleen is very frequent. Persistent anemias, which do not yield to treatment, frequently follow malarial fever.

The *diagnosis* of malarial fever in reality depends upon demonstrating by the aid of the microscope the malarial parasite in the blood. The clinical symptoms, however, in most cases of malaria are so characteristic that a clinical diagnosis usually can be made. However, no diagnosis should be recorded as malaria unless it has been definitely confirmed by microscopic methods. The fact that malarial parasites cannot always be found in the peripheral blood, however, does not necessarily show that the diagnosis of malaria can be dismissed. Splenic puncture will often yield blood specimens in which the malarial parasite can be demonstrated. The periodicity which occurs in the fever curve in the various forms of malarial fever is most characteristic, and usually will lead to diagnosis. However, marked temperature intermissions or remissions due to accumulations of pus, especially when these occur in the liver, must be carefully excluded. It is often stated that a fever that is not influenced by four days of active quinin treatment may be dismissed as malaria. This is usually true with regard to fevers due to the tertian and the quartan parasites, but may not be true in fevers caused by the subtertian parasites. It is not an infrequent experience to find a fever unaffected by quinin, and yet to find malarial parasites of the subtertian variety in the peripheral blood. In malarial cachexia a careful clinical examination, especially hourly temperature charts, may be of more value in arriving at a correct diagnosis than blood examinations. Repeated examinations of the blood, however, will often result in demonstrating the malarial parasite in the erythrocytes, or finding hemozoin in the white corpuscles. The differentiation of bilious remittent fever and yellow fever at times causes considerable difficulty. When due to malaria, albuminuria is not common, and is seldom ever so marked as in yellow fever. The temperature in malaria may remain continually high, while in yellow fever there is a sharp remission at the end of three or four days. There is more vomiting in yellow fever. The pulse does not

become slow in malarial fever. Cerebrospinal meningitis at times offers difficulties, but the rigidity of the muscles of the neck usually leads to blood examinations which make it possible to exclude malaria. With the aid of the microscope there is usually no difficulty in separating malarial fever from many of the diseases which cause chills and fever, as, for instance, urethral fever, the passage of gall-stones, pyelitis, lymphangitis, especially that associated with elephantiasis and filaria, Mediterranean fever, kala-azar, ulcerative endocarditis, typhoid fever, abscess of the liver, rapidly growing sarcoma, visceral syphilis, and many obscure and ill-defined conditions.

The *prognosis* of malarial fever is closely associated with climate, race, age, sex and type of parasite causing the infection, and the presence or absence of organic complications. An idiosyncrasy against quinin is also at times an important factor. With regard to climate, it is well known that recovery does not take place so readily when the stimulating effects of cool air are not available. In persons from the temperate zone, who later contract malarial fever in tropical countries, the prognosis is not as good as in those who have passed their childhood in malarial districts. During childhood a certain amount of immunity is apparently acquired by the infections which usually take place. Infection with tertian and quartan parasites affords a much better prognosis than infection with the subtertian. The pernicious forms and latent malarias, as well as malarial cachexia, do not always furnish a favorable prognosis, yet if an accurate diagnosis is made reasonably early and experienced treatment is available, there is considerable hope that complete elimination of the infection can be brought about. Persons who are unable to stand usual doses of quinin should not risk residence in malarious countries. The mortality among Europeans in places where pernicious forms of malaria prevail is usually high. It is stated that the mortality among natives of such districts is low, but this view may be subject to correction under more careful observation.

Malarial fever is no doubt frequently confounded with ankylostomiasis, kala-azar, typhoid fever, and similar conditions which cause chills and fever.

TREATMENT.

It is generally stated that the use of quinin in malaria is one of the few instances of a true specific which occurs in medicine. This statement seems to be true in so far as the great majority of the cases are concerned, but there can be little doubt that there are many cases of malarial infection in which the use of quinin, given under the most favorable circumstances, fails completely to eliminate the infection from the human system. Recent developments in the study of malaria show that the treatment of the disease by quinin, as ordinarily administered, is most ineffective. Many thousands of individuals who are regarded as cured of malarial fever after a few doses of quinin, still have latent infections which at any time may prove detrimental to the individual, and act as reservoirs from which malarial infection is distributed far and wide. The greatest difference of opinion prevails among medical men of large experience as to the best salt of quinin to employ, the dose, and the intervals at which it should be administered. Many of these differences, in all probability, arise from the fact that some persons apparently do not absorb quinin through the intestinal tract, but when administered hypodermically a favorable result is obtained. It often happens that an individual will not absorb the drug through either of these channels, when the intravenous method often furnishes a solution. Many salts of quinin have been advocated from time to time, but sulphate of quinin seems to be more generally used than any other salt, and when administered, so far as the oral route is concerned, in an intelligent manner, probably gives as good average results as many of the other salts or the fanciful preparations which have been placed upon the market. When quinin is administered by mouth it probably gives the best results in the liquid form, but owing to the bitterness of the solution it is practically impossible to give quinin in this way. For administration per oram the two methods of choice usually narrow down to pills and capsules. Pills very often have a sugar or other coating which is not readily dissolved by the gastric secretions, and are passed through the system without any absorption having taken place. If it is necessary to use pills, an

investigation should always be made to ascertain whether they are being absorbed. Probably one of the most safe and satisfactory methods of giving quinin is by the means of capsules. If there is any doubt as to their absorption, small doses of water acidulated with hydrochloric acid may be administered immediately after the quinin has been taken.

There have been many reasons advanced for the administration of quinin at certain intervals. The most universal custom has been to administer the drug three times a day after meals. Recently Ochsner[11] drew attention to his experiences in the treatment of malaria in Mexico, for which he claims unusually satisfactory results by the administration of 2 grains (0.13 Gm.) of quinin at two-hour intervals, followed by withdrawal of the drug for five days, and then a repetition of the dose. (For complete details of this treatment see method of administration below.) This mode of administration is based on the theory that quinin will kill adult malarial organisms, but will not kill sporozoïtes. Consequently, if quinin is given continuously the development of the sporozoïtes is retarded, but they will grow into adult forms as soon as the quinin is withdrawn. By allowing a suitable interval between the quinin treatments an opportunity is afforded for the sporozoïtes to develop into adults, and they can then be effectively killed by the subsequent administration of quinin.

It has been customary in the past to administer to robust adults 10 grains (0.65 Gm.) of quinin in two 5-grain (0.32 Gm.) capsules, given three times daily, or a total dose of 30 grains (1.9 Gm.) every twenty-four hours. Work recently undertaken in the Southern States by Bass in the treatment of malaria indicates that among persons who are chronic carriers a dose of 10 grains (0.65 Gm.) per day is insufficient properly to sterilize the blood. In order to make treatment effective administration of quinin should be followed at subsequent intervals by careful examinations of the blood to ascertain whether any parasites are present, and quinin should be repeated in all cases where they are found.

Until quite recently many of the older physicians recommended the use of Warburg's tincture after quinin given in the ordinary form had failed, but it is now considered to be

of doubtful value, and not nearly so efficient in obstinate cases as the administration of quinin by the intravenous, or even the hypodermic, method. Quinin in large doses is a cardiac depressant, and this fact must be borne in mind when administering the drug to the old and the feeble, especially by intravenous or intramuscular injections. Quinin acts as a stimulant to the nervous system, and is said to cause an increased flow of blood to the brain. In the tissues it is partly destroyed by oxidation, and usually the whole quantity administered can be found in the urine. Excretion takes place through the kidneys in the form of quinin dihydroxyl. When given in large doses it causes cerebral congestion, which produces buzzing in the ears, headache and deafness, due to congestion of the middle ear. These symptoms can usually be greatly relieved by bromids, or by ergot. Quinin eruptions occur from time to time in the form of erythematous, or even papular, vesicular, and urticarial eruptions. The belief is very generally held that the administration of quinin in some persons may cause hemoglobinuria. Amblyopia is another untoward result, and appears to be due to the contractions caused in the retinal arteries, which may even go on to degeneration of the retinal ganglion, and cause serious permanent eye disturbances. Quinin is a stimulant to the uterus, and its use in pregnancy must be carefully guarded. In mild quartan and tertian fevers there is little doubt that excellent results are obtained by administering quinin four hours before the attack. This corresponds to the period of the sporulation of the parasite. As stated before, however, the administration of the drug three times a day, without fine theoretical considerations as to its effect upon parasitic life, apparently produces results which are just as successful as any other kinds of administration which have been suggested.

Method of Administration. In the past it has been customary to treat all ordinary cases of quartan and tertian fevers with 10 grains (0.65 Gm.) of quinin sulphate, given in 5-grain (0.325 Gm.) capsules, three times a day with meals. Much has been written about the desirability of employing quinin salts that are readily soluble in water, but in actual experience quinin sulphate appears to be as effective when

given by mouth as salts, which are more soluble. Quinin bisulphate is probably the most soluble, and the best drug to use, and it may be administered in the same doses as the sulphate. Ochsner's[12] method consists in an exclusive diet of hot soup for ten days. On the evening of the first day 2 ounces (60 mils) of castor oil are administered. At 6 A.M. on the second day he begins by giving a 2-grain (0.13 Gm.) capsule of quinin, preferably bisulphate, with the cover of the capsule removed, with ½ pint (236 mils) of hot water, every two hours night and day for 30 doses. He lays great stress upon not missing a single dose, owing to the importance of keeping fresh quinin constantly in the blood. This is followed for six nights and five days with a pill of $\frac{1}{50}$ grain (0.00130 Gm.) of arsenous acid with ½ pint (236 mils) of hot water at 6, 9, 12, 3 and 6 o'clock. At the end of this period castor oil is given as on the first day. At 6 A.M., following the sixth night, he again repeats the 2-grain (0.13 Gm.) doses of quinin at two-hour intervals until 30 doses have been taken. This is followed by general tonics and wholesome nourishing food. In pernicious forms of malaria where the prompt exhibition of quinin is essential to save life, the bihydrochlorid should be given intravenously in 5-grain (0.324 Gm.) doses. The technic should be the same as that employed in the administration of salvarsan.

If the case is particularly aggravated, this treatment should be repeated at intervals of several hours, and the dose increased if the circumstances seem to demand it. In very serious cases as much as 40 grains (2.6 Gms.) of quinin may be given intravenously during twenty-four hours. In ordinary cases of malaria, and especially the chronic type which do not respond readily to treatment, better results frequently can be obtained by administering, in connection with the quinin, 3 mils (48.6 *m.*) of fluidextract of ergot. The action of the ergot in this connection has been explained on the basis that ergot being a stimulant of non-striped muscular tissue causes contractions in the spleen and small blocked blood-vessels which result in forcing malarial parasites into the general circulation where they can readily be reached by the quinin. This explanation seems rather doubtful, but it has been frequently demonstrated that ergot is efficient in connection with quinin

in certain types of malaria. In latent or chronic malaria good results may be obtained, after quinin has failed, with 10-grain intravenous doses of salvarsan or neosalvarsan. In chronic malaria, or cases in which there has been extreme prostration, or in which quinin does not seem to be effective, Fowler's solution in ascending doses often brings about prompt improvement. It is well to begin with 3-drop (0.18 mil) doses in water three times a day, with an increase in the total dose for the day of 1 drop until the point of tolerance is reached. This usually happens before 8 drops (0.50 mil) per dose, or a total of 24 drops (1.50 mils) for the day, is being taken. It is then well to reduce the dose by 2 drops (0.12 mil). That is to say, if 8-drop (0.50 mil) doses are being given with a total of 24 drops (1.50 mils) per day, 6-drop (0.36 mil) doses should be given with a total of 18 drops (1.12 mils) per day. As a rule, hypodermic injections of quinin are not to be recommended. Much better results are obtained, and the danger from abscess formation is greatly reduced, if intramuscular injections are used instead. For this purpose the bihydrochlorid is the best salt to employ. Baccelli's formula, which is frequently used for this purpose, consists of 10 grams (154.3 grs.) of bihydrochlorid of quinin and 0.075 grams (1.1 grs.) of ordinary salt dissolved in 10 grams (154.3 grs.) of water. One-tenth of this mixture is used for each injection. Great precaution must be observed to ensure the sterility of the mixture. Injections of quinin are frequently associated with abscess formation, and this must be rigidly guarded against. In malarious countries, in which intramuscular injections of quinin may be frequently employed, it is well to keep a quinin solution in sterilized tubes of the proper dosage. A preparation suitable for this purpose can be made with 10 grams (154.3 grs.) of bihydrochlorid of quinin, 18 grams (277.7 grs.) of distilled water, 5 grams (77.1 grs.) of ethylurethane. Of this quantity one-twenty-fifth portion is used for an injection. One and a half mils (25 *m.*) of the solution contain five-tenths of a gram (7.71 grs.) of quinin. This solution is often referred to as Gaglio's or Giemsa's. Where it is possible to give quinin by mouth, this may be done with suitable doses of quinin sulphate, to which 5 to 10 minims (0.30 to 0.60 mils)

of dilute sulphuric acid have been added, with syrup of orange and distilled water. The prescription per dose is as follows:

℞ Quinin sulphate ..... .65 (10 gr.).
Sulphuric acid dilute ..... .12 (10 *m*).
Syrup of orange ..... 4 mils (64.8 *m*).
Distilled water .....q. s. 25 mils (6.7 f℥).

It is generally held that quinin absorbs much more rapidly when administered in an acid solution.

During the chilly stage patients can usually be made very comfortable and the chill promptly stopped by the injection of 10 drops (0.62 mils) of chloroform. If this fails immediately to relieve the chill, morphin sulphate in ⅙-grain (0.01 Gm.) doses, given hypodermically, generally brings relief. The headache is greatly relieved by cold applications. When ice is not available, cloths may be soaked in a mixture composed of salt, fresh limes or lemons, with vinegar or weak acetic acid, and some eau-de-Cologne. These have a very cooling effect, and should be changed as frequently as they become warm. Acetanilid and caffein also may be employed to relieve the severe headache. Vomiting frequently can be relieved by sips of iced soda-water or champagne. If the vomiting is severe and not relieved by the foregoing, a small mustard, or, preferably, a capsicum plaster may be applied to the pit of the stomach. Gastric lavage may be undertaken in severe cases to relieve the vomiting. The treatment of malaria should invariably be begun by ¼-grain (0.01 Gm.) doses of calomel, given at hourly intervals until free purgation takes place. Many practitioners follow calomel with suitable doses of magnesium sulphate, but experience shows that this is scarcely necessary, and only makes the patient more uncomfortable. The usual liquid diet should be given during the fever periods, and even during the intervals of apyrexia it is well to allow only easily digested foods. Meat, as a rule, is borne badly. In chronic malaria, after regular quinin treatment, a change of climate is most desirable and usually necessary, if prompt permanent convalescence is to be established. A tonic should be given as soon as convalescence begins. The U. S. P. preparation of the elixir of iron, quinin and strychnin, is among the best that can be employed.

Prophylaxis. Malarial fever, especially the appearance of new cases, depends upon the exposure of numerous human beings infected with male and female gametocytes to *Anopheles* mosquitoes in which gametocytes are capable of developing into sporozoïtes, and suitable temperature conditions for the development of the parasite in the mosquito. Much success has been had in bringing malarial fever under control in various countries of the world. For instance, Ross achieved great success in Ismailia; in Panama excellent results were obtained, likewise in the Philippines, and more recently in the Federated Malay States. Unfortunately the cost of these mosquito-control measures has been greater than the funds which could be made available in the average district. In other words, the per capita taxpaying power of the average community is not sufficiently great to carry out malarial measures that are as expensive as those cited above. It is true that considerable success has been had on a small scale in bringing malaria under control in many parts of the world at a cost which was reasonable, but these are only isolated instances, and there is as yet no feasible plan for bringing about the control of malarial fever through the elimination of mosquitoes at a cost which would make its adoption universally practicable.

From the foregoing it will be apparent that malaria is a controllable disease. From the theoretical standpoint control is simple. The malarial parasite has two hosts, man and certain mosquitoes belonging to the *Anophelinæ* group. Both of these hosts are necessary to the perpetuation of the parasite and of the disease, so that theoretically it is only necessary to break a link in this chain. Every infected person has derived his infection from an infected mosquito, which in turn, has derived its infection from an infected person, so that if blood of man can be sterilized, so far as the malarial parasite is concerned, a mosquito would have no place from which to derive its infection. On the other hand, if mosquitoes could be eliminated, there would be no means to convey the infection from one human being to another, and the disease would automatically stop. At the present time a number of experiments are being conducted in the Southern States, with a view to determining exactly the per capita cost of eradicating

malaria under different circumstances and under different methods. One of these consists in taking a selected community and making a blood examination in the spring of all the persons before the mosquitoes become active, and in treating with quinin those found infected. The other experiment consists in attempting to eliminate *Anopheles* mosquito breeding-places from the vicinity of human habitations by simple drainage or with larvæ-destroying agents. None of these experiments has yet been completed, but experience with the first experiment shows that persons who are carriers of malaria cannot be sterilized by the administration of quinin in 10-grain (0.65 Gm.) daily doses over a period of several weeks. Even 20-grain (1.3 Gms.) doses given over a period of several weeks do not cause a complete disappearance of the malarial parasite in every individual. A similar experiment, but not upon so intensive a plan, has been carried out in Italy. It has been shown, for instance, in certain districts of Italy that the number of malarial deaths seems to be in direct proportion to the quantity of quinin sold. For instance, in 1901 there were 13,861 deaths recorded in Italy. In 1902, 2242 kilos of quinin were sold, and the deaths dropped to 9908. In 1906, 20,723 kilos of quinin were sold, and the deaths dropped to 4871. In 1900 Koch conducted a similar experiment in New Guinea, but upon a very much smaller scale. Among 157 persons who harbored the malarial parasite he administered 1 gram (15.4 grs.) of quinin every eighth or ninth day until the malarial parasite disappeared. The number of hospital admissions was reduced in a period of six months from twenty-four to one. A similar experiment was made by Heiser in the Philippines at the Iwahig Penal Colony, where the precautions were adequate to insure that the quinin actually reached the stomach. He administered 5-grain (0.32 Gm.) doses of quinin to 1000 prisoners daily. In addition, the use of mosquito-nets was insisted upon. There was an enormous reduction in the number of new cases, but after a year's trial it became evident that the quinin administered did not give complete protection, and that, perhaps, the results obtained were also largely contributed to by the strict use of mosquito-nets. It was found, for instance, that when prisoners who were taking the regular doses of quinin slept in

camps in the jungle and were not protected by the mosquito-nets, they almost invariably contracted malarial fever. This experiment was also interesting, owing to the fact that there were no persons residing within the flight of the mosquitoes who did not receive daily doses of quinin. Any measure which gives an individual protection from mosquitoes at night is more than likely to reduce the incidence of malarial fever. Recent observations made in Sandakan, Borneo, showed that in a school which was attended by students who lived at the school and those who attended only during the day, the infection among those who slept in the school was very much heavier than those who slept outside. It was shown that the pupils in the school lived in a district in which the *Anophcles* mosquito bred freely, and they were not protected by mosquito-nets, whereas the day pupils lived in their own homes in districts comparatively free from mosquitoes and slept under mosquito-nets.

It is obvious from what has been stated above that malarial control in the future must largely depend upon the wide dissemination of knowledge with regard to the manner in which the disease is spread. The best hope of bringing this about would seem to be to have the subject thoroughly taught in the public schools, and also to have public lectures with lantern demonstrations for adults.

For persons who are compelled to go temporarily into malarious districts reasonable protection against infection may be had by the administration of 10-grain (0.65 Gm.) daily doses of quinin and the rigid use of mosquito-nets. Koch thought 1 gram (15.4 grs.) once a week was sufficient. Whether persons who actually live or make long sojourns in malarious districts can take sufficiently large daily doses of quinin to give themselves protection is an open question. The best hope of success for such persons would seem to lie in an attempt to prevent mosquitoes breeding in the neighborhood of their habitations, to use mosquito-nets, and to have their living quarters thoroughly screened against mosquitoes.

The Anopheles Mosquito. Experience so far has shown that in order to bring malarial fever under control it is not necessary to bring about complete eradication. If, for in-

stance, the mosquito index for a community is 100 per cent., if a 90 per cent. reduction is made in the number of mosquitoes, it is more than likely that malaria will disappear. This is a most important observation, and has great practical value. In many communities it is possible to bring about a great reduction in mosquitoes, but it is not possible to eradicate them completely. In general, it may be stated that if a 50 per cent. reduction can be brought about in the number of mosquitoes, the results in a reduced number of malarial cases will be in proportion to the percentage which is reached, about 50 per cent. The practical measures for eliminating mosquitoes may depend upon drainage, and upon the spreading over the water surfaces some larvicide, as, for instance, kerosene oil. Experience in the Philippines has shown that a desirable mixture can be made with ordinary cheap kerosene and crude petroleum in equal parts. "Larvacide," which is a preparation successfully used in Panama, is made as follows:

Carbolic acid crude	150 gals.	(600 l.).
Rosin	20 lbs.	(9.07 Kg.).
Caustic soda	30 lbs.	(13.60 Kg.).
Water	6 gals.	(24 l.).

This solution actually mixes with the water in which it is placed, and is very destructive to larvicidal growth, but it also destroys plant life, and should be used with caution where this is objectionable. Oiling of water surfaces should be done at intervals of at least every ten days, because an adult mosquito, under favorable conditions, may develop in eleven to twelve days after the deposition of the egg on the surface of the water.

Briefly, then, the whole question of prophylaxis may be summed up by the statement that up to the present time no definite conclusions have been reached as to the best and most economical methods to employ for a given set of conditions. Each case must be decided upon whether it would be better to use a combination of the quinin, mosquito-net and mosquito-destruction measures, or whether a single measure or more should be used.

YELLOW FEVER.

Yellow fever is an acute febrile disease of unknown origin, characterized, as a rule, by two paroxysms of fever, which are separated by a remission or an intermission, accompanied with albuminuria, jaundice, hemorrhages from the stomach, and no particular change in the leucocyte count. It is transmitted by the *Stegomyia calopus* mosquito, heretofore known as the *Stegomyia fasciata.*

Pestis Americana, Typhus Icteroides, Fibre Amarilla, Magdalena Fever (Columbia), Pest of Havana (Cuba), The Yellow Jack, are synonyms commonly used in referring to this infection.

The origin of the disease is shrouded in considerable mystery. It has been reported as having been originally a disease of the Antilles, and to have attacked the troops of Christopher Columbus in 1495 in Santo Domingo, whence it was carried to the mainland of America. The endemic home of the disease is at present in or near Guayaquil, and in the vicinity of Bahia. In the past few years it has been frequently carried to Central and other South American countries, but apparently has gained no permanent foothold. As a result of the efforts of American sanitarians it has disappeared completely from Cuba and Panama. Similar work in Rio de Janeiro has produced results almost as good. The disease frequently is reported as occurring on the Gold Coast of Western Africa, but the diagnosis of yellow fever is not universally accepted. In the past it has been carried to Spain and Italy, but it never gained permanent foothold in these countries. Until comparatively recent times it was frequently carried to the ports of the Atlantic seaboard of America south of Baltimore and along the Gulf of Mexico. During the early part of the nineteenth century the disease appeared as far north as Philadelphia.

In 1881, Charles Finley, of Havana, formulated the hypothesis that the spread of yellow fever was through the mosquito. In 1882 Gererd permitted himself to be bitten by a mosquito which had fed on a yellow fever patient on the fourth day of the disease, and he in turn contracted an attack of yellow fever. In 1897 Sanarelli announced that he had found the bacterium of yellow fever, which he named the

Bacillus icteroides. These findings were confirmed by other observers. In 1900 Reed and Carroll announced that the *Bacillus icteroides* belonged to the hog-cholera group, and was probably identical with the *Bacillus choleræ suis.* Numerous other workers have reported on the causative agent, but it may be stated that none of these various organisms have withstood the test of time, and the true etiology of the disease is still unknown. In 1900 Reed, Carroll, Agramonte and Lazear proved that the disease could be produced by the subcutaneous injection of infected blood into a non-immune person; also that the disease was not contagious, and that it was only spread by the bite of the *Stegomyia calopus.* Their work was speedily confirmed by many workers of numerous nationalities. In 1909 Seidelin described the presence of minute bodies, which he called *Paraplasma flavigenum,* in the red cells of persons suffering from yellow fever. This, however, has not been very generally confirmed. The theory of the propagation of yellow fever by the *Stegomyia calopus* has been tested practically with good results in Havana, Texas, Mexico, New Orleans, Panama and Rio de Janeiro. The discovery of the means of transmission of yellow fever by the mosquito was one of the first important American achievements in tropical medicine. It should also be mentioned that Lazear sacrificed his life in permitting the yellow fever mosquitoes to bite him.

In brief, it may be stated that yellow fever is only possible in those regions of the world in which the *calopus* mosquito thrives. With the elimination of this mosquito, the disappearance of the disease invariably results. An interesting phenomenon is the fact that the eradication of yellow fever does not depend upon the elimination of the *Stegomyia calopus.* Judging by the experiences in Havana and Rio de Janeiro, it seems quite likely that if a 90 per cent. reduction in the number of mosquitoes can be effected, the disease will disappear. The disease may spread through the agency of ships or other methods of transportation which are capable of conveying *Stegomyia calopus* from areas in which yellow fever prevails. The opportunities for spreading the disease into far removed countries is immeasurably increased by the presence on board vessels of persons who are afflicted with

the disease who may come in contact with *Stegomyia* mosquitoes on the ship or on shore. It appears that frost, which results in the destruction of mosquitoes, invariably brings an outbreak of yellow fever to a close.

That the infection of yellow fever exists in the blood can readily be proven by the fact that subcutaneous inoculation of 0.12 of a mil (*2 m.*) of infected blood into a non-immune person will produce an attack of the disease, provided that the blood has been drawn from an infected individual during the first three days of illness. It is not likely to be transmitted to man by post-mortem wounds, because death usually does not take place until after the third day of the illness, when the patient is no longer in an infective state. For the transmission of the disease by mosquitoes, it is necessary for the mosquitoes to bite the individual during the first three days of the disease, and then twelve days must elapse before the mosquito is capable of transmitting the infection. The proof of the transmission of the disease by the *Stegomyia calopus* mosquito is furnished by Reed,[13] Carroll,[14] Agramonte[15] and Lazear,[16] through the construction of a mosquito-proof building, which was divided into two compartments, in the first of which infected mosquitoes were liberated and allowed to bite a non-immune, while in the second compartment non-immunes slept. The man in the first compartment developed an attack of yellow fever, while the others did not. Further proof came from the fact that non-immunes who lived in mosquito-proof houses, with articles of clothing and bedding soiled with the discharges of yellow fever cases, did not contract the disease; while non-immunes who were kept free from such infection, but exposed to mosquitoes that had bitten yellow fever cases, did contract the disease.

The experiments of Marchoux and Simond[17] seem to indicate that the infection can be transmitted through mosquito eggs, and thus a second generation of mosquitoes may be transmitters of the disease. It is also more than likely that the only reservoir for the disease is the human and the mosquito. Apparently chimpanzees have been infected, but it is unlikely that they are an important factor in the transmission of the disease to man.

There are usually numerous changes in the liver. The

cells swell and press upon the bile capillaries, and thus obstruct the flow of bile and cause hepatogenous jaundice. This swelling also blocks the intralobular capillaries, which causes congestion in the viscera drained by the portal circulation. The liver cells usually degenerate so extensively as to cause a lessening in the urea function, and often a condition of ammoniemia may occur, from which serious toxic effects in the brain and other organs are to be expected. The disease often seriously affects the endothelial lining of the blood capillaries, and causes hemorrhages in various parts of the body. The attack usually confers a permanent immunity. In countries in which yellow fever is epidemic it is more than likely that children have mild attacks which go unnoticed, and they therefore become immune. In yellow fever countries it is very rare to find that the disease occurs among persons who have been many years resident. It is usually the newcomers who are affected.

There may be a slight increase in the number of leucocytes, but this is not usual. In brief, it may be stated that few changes are to be looked for in the blood.

Albumin appears early, very often on the second day, and increases in quantity, especially in severe cases. This symptom, in combination with the fever and jaundice, is usually greatly depended upon for a diagnosis. Bile is usually present in the urine on the fifth or sixth day; and casts are the rule, first hyaline, then granular, and finally epithelial. The amount of urea is greatly diminished, and the quantity of urine is scanty.

The material vomited by the patient is usually referred to as black vomit, since it consists largely of blood and mucus. In some instances the vomit may be red, in which event it is composed of bright blood which has not had an opportunity to coagulate. Microscopically, the vomit is found to consist of red blood-corpuscles, epithelial cells, *débris* and micro-organisms.

The skin is bile-stained and blotched with *post-mortem* lividity and hemorrhages. The liver is yellowish in color and hemorrhagic, with the cells swollen and in a state of advanced fatty degeneration. The gall-bladder contains thickened bile, which is sometimes mixed with blood. The spleen is normal

in size. The stomach and intestines are often filled with blood. The kidneys, normal in size and frequently congested, as a rule, show evidences of acute nephritis. The cells of the tubules show fatty degeneration, and the lumen may contain granular *débris*. The bladder is usually empty. The serous surfaces of the heart frequently show punctiform hemorrhages, and effusions may occur in the pericardium. The lungs are congested, and hemorrhages are often found between the pleura. The uterine mucosa is congested, and there may be blood in the canal. The meninges of the brain are congested and hemorrhagic areas are frequent.

The *incubation period* of yellow fever may be safely placed between two and six days, and this premise is an important factor in determining the diagnosis. The average period of incubation appears to be about five days. The attack usually begins with headache, flushed face, injected eyes, and pains in the body, particularly in the back. Albuminuria appears on the second day. The temperature may be divided into two paroxysms. The first lasts for from two to four days, after which the fever usually subsides to normal, accompanied by sweating. In some instances it only remits to about 100° F. (37.7° C.), but the symptoms largely disappear. At the end of this first paroxysm convalescence may begin. When this does not occur after a period of a day there is a sharp rise in the temperature, which is not accompanied by a corresponding increase in the pulse rate. As jaundice appears at about this time, this is naturally to be expected. However, it is of the greatest diagnostic importance. The jaundice which accompanies the second paroxysm, plus the fever, gives the name Yellow Fever to the disease. The appetite is lost from the beginning, and there is usually vomiting, associated with pain and tenderness over the gastric region. Constipation is the rule. The urine is high colored, decreased in amount, with acid reaction, and high specific gravity. Insomnia during the early state of the disease is frequent. During the secondary rise of temperature all the symptoms of the first paroxysm return, especially the vomiting and tenderness in the gastric region. Hemorrhages may occur from the nose, mouth or uterus, and black vomit is common. Albumin increases rapidly. In severe cases there may be

complete suppression of urine. Delirium is common, accompanied with much restlessness. In cases in which there is a favorable termination, after the third day there is usually an increase of the amount of urine, a decrease of the albumin, and then vomiting gradually ceases; the patient passes into deep sleep, and is then well on the road to convalescence. If the outcome is fatal, the temperature continues, the jaundice deepens, hemorrhages appear under the skin, and there are hiccough, subsultus tendinum, clammy sweats, complete suppression of the urine, coma, and convulsions, which lead to death. Death, however, may take place before respiratory and cardiac failures can occur.

Yellow fever usually is described by different authors as occurring in a number of types. It is most difficult to draw any sharp distinction between the different types. In general, the cases are either mild, severe or malignant, with symptoms that correspond to these conditions. The malignant type very often begins with a fever of 105° to 107° F. (40.5° to 41.6° C.), with violent vomiting and the early appearance of black vomit, and the patient dies during the first paroxysm of fever without any remission in the temperature having taken place.

Those who have any pathologic condition usually resist yellow fever badly, while those who are in robust health show the greatest percentage of recoveries. Renal and cardiac affections are particularly unfavorable complications, and usually persons afflicted with them succumb to the disease.

The convalescence, as a rule, is neither complicated nor protracted, but at times boils, abscesses, dysentery and inflammation of the liver may occur.

The mortality varies very greatly in different outbreaks. There have been mild outbreaks in which the mortality has been only about 10 per cent.; whereas in the United States it sometimes rises to 25 per cent., and in West Africa from 45 per cent. to 80 per cent. Yellow fever must be regarded as a serious disease, especially if complicated with some pre-existing disorder.

During an epidemic the typical symptoms enumerated above are usually sufficient to establish a diagnosis, but the first cases, and especially mild attacks of the disease, are most difficult to recognize. Among the best signs for dependable

diagnosis are the early albuminuria, the gastric tenderness, the jaundice, the secondary rise in temperature and the black vomit. The disease usually has to be differentiated from dengue, subtertian malaria, blackwater fever and relapsing fever. Dengue may be recognized by the absence of marked albuminuria and jaundice; subtertian malaria by the parasites in the blood; blackwater fever by the presence of hemoglobin in the urine and the increase in the mononuclear lymphocytes; and relapsing fever by the parasites in the blood and the leucocytosis.

TREATMENT.

As the cause of the disease is not known, the best hope of success is directed toward a rapid elimination of the toxins, and bringing comfort to the patient. An important consideration is the prophylaxis of the disease. This is best accomplished by effectively screening the patient from mosquitoes and the destruction of those mosquitoes in the house which may have bitten the patient. The treatment must be directed mainly toward stimulation of the organs of elimination, that is, the bowels, skin and kidneys. The bowels are to be opened by small doses of calomel, followed by magnesia or sodium sulphate, until active purgation has been produced. The free action of the bowels can be continued by the administration of tablespoonful doses of sodium sulphate dissolved in a pint of water and given morning and night. Hot blankets and hot-water bottles promote the free action of the skin. In order to dilute the toxins and to stimulate action of the kidneys, plenty of alkaline fluids are desirable. These can be supplied by the use of from 2 to 4 quarts (liters) of Vichy or Poland water, or the same quantity of any reliable alkaline mineral water. If none of these are available a substitute may be made by adding 30 grains (1.95 Gms.) of sodium bicarbonate to a pint of water. An effervescing drink can be made by the use of sodium bicarbonate and fresh lime-juice. Care should be taken to use the mixture in neutral quantities. This, if necessary, may be brought about by the use of sodium sulphate. If fluids cannot be given by the mouth, enemata should be used. The headache and pains in the back muscles may be relieved by 3-grain (0.195 Gm.) doses of acetanilid.

The severe pains in the back may be further relieved by hot applications. Vomiting may be controlled by the use of iced champagne, and at times by counter-irritation in the form of a blister over the stomach. The fever may be reduced by cold sponging. Vomiting sometimes may be checked by the use of the ice-bag over the stomach. The weakening heart must be supported by injections of strychnin, followed, for more permanent effect, by small doses of digitalin. Camphor is also valuable. Milk with lime and barley-water is the dietary mainstay. Ice-cream, and wine and lemon jellies, are also desirable. The return to solid food after the absence of high temperature should not be undertaken until at least three days have elapsed. Solid food seems to be very badly borne by those who have passed through a severe attack of yellow fever. Death may occur from overeating during convalescence.

Prophylaxis. To prevent the spread of yellow fever it is only necessary to prevent mosquitoes which have bitten infected individuals between the second and fifth day of the disease from reaching other individuals twelve days or more afterward. The patient should be kept under a mosquito-net. The house should be thoroughly screened. In all places where yellow fever has appeared fumigation of the premises should be carried out, in order to kill any possible mosquitoes. This can be done by burning 2 pounds of sulphur for each thousand cubic feet of air space. The sulphur is best burned in an iron pot, which has been set in a receptacle containing water. The doors and windows must be closed and cracks sealed by the means of pasted paper. Three hours is ample time for the fumes to act. If sulphur cannot be used, pyrethrum powder, or, as it is sometimes called, Japanese powder, may be used in the proportion of 2 pounds (0.9 Kg.) to each thousand cubic feet of air space. However, attention should be drawn to the fact that pyrethrum gases do not kill mosquitoes. They are only numbed, and immediately after the burning has been completed the room should be swept and the collected mosquitoes burned. Quarantine against residents of yellow fever districts is unnecessary, if such persons can be kept free from the bites of *Stegomyia* mosquitoes. Ships that have anchored in yellow fever ports should be

fumigated against mosquitoes after exposure. Attendants at quarantine stations should live in mosquito-proof houses, so that possible infection among them may not spread to mosquitoes. Mosquitoes that have sucked blood from dead bodies seldom are infective, because patients usually do not die before the fifth day of the disease, and their blood is no longer infective at that period of the disease. Screens against mosquitoes should contain eighteen meshes to the inch (2.5 cm.), if made of fine wire, or a correspondingly smaller number of meshes if the wire is of a heavy variety.

DENGUE.

There are many synonyms for this disease. A popular name in Texas for dengue is Breakbone Fever, and in other parts of the world it is variously referred to as Breakheart Fever, Giraffe Fever, Febris Endemica cum Roseola and Seven Days' Fever.

Dengue is an acute, specific fever of unknown origin, usually characterized by two febrile paroxysms, separated by an intermission, by albuminuria and by marked leucopenia. Most authorities attribute its transmission to the *Culex fatigans* mosquito. Recent work (1916) in Australia indicates that it also may be spread by the *Stegomyia fasciata.*

It appears to have been recognized in Java in the eighteenth century by Bylon, but there are old descriptions of disease which indicate that it was reported even at an earlier period in Seville. In general, it may be stated to exist in those areas of the world in which yellow fever has occurred, or in which the *Stegomyia fasciata* mosquito is common. There are frequent outbreaks in Texas. It is a very disabling disease in Australia, and in cities like Brisbane, for instance, the entire population may be affected before an outbreak comes to a close. Constant efforts have been made throughout the world to find the causative organism, but so far without success.

Graham,[18] Ashburn and Craig[19] have done the work which is generally considered as throwing most light upon its transmission. Their observations lead to the conclusion that the disease is intimately associated with mosquitoes. The intra-

venous injection of dengue-fever blood produces typical attacks of the disease in healthy subjects within two or three days. There is no evidence of the spread of the disease other than through mosquitoes. As there is practically no mortality from dengue, little or nothing is known of the pathology of the affection. The lesions in persons who have died of some intercurrent affection while suffering with dengue do not show anything characteristic beyond the lesion of the intercurrent affection.

Based on the observations of Ashburn and Craig, the *incubation period* is from three to six days. Some authors report that prodromata may appear several days before the outbreak of the disease, but this is unusual. The attack is generally sudden, and begins with severe pain in some part of the body. Often there is a sensation of extreme fatigue, and chills may occur at times. A characteristic onset is with great pain behind the eyeballs and intense headache. Another very common symptom is great pain in the back, particularly in the sacroiliac joint. In children the onset may be characterized by convulsions or delirium. The temperature rises rapidly, and usually is between 103° and 106° F. (39.5° and 41.1° C.). The pulse usually increases in proportion, and is found to be from 90 to 140 per minute. Very frequently the joints are painful. As a rule, the joints can be moved passively without causing pain, but if there is active movement the pain is severe. In rare instances there is redness and swelling in the joints. The general character of the pains aptly may be described by a quotation from Rip Van Winkle: "Every time I make a new move I have a new pain." There may be either constipation or diarrhea. The glands are not enlarged. Leucopenia is so constant that it is of great diagnostic importance, and the leucocyte count varies from 5000 to 1000, the average being about 3500 per cubic millimeter. The leucocytes are normal in appearance, but there is a marked increase in the number of smaller lymphocytes, and a commensurate loss of the polynuclear forms. The urine is highly colored, and frequently contains traces of albumin. On the third day the temperature falls rapidly, and is often accompanied by profuse perspiration, the passage of much urine, violent diarrhea and bleeding from the nose,

These signs, however, may be absent. The patient feels much better. This intermission, however, may be entirely lacking. When it does occur it lasts usually until the fifth day, when there is a rapid rise in the temperature, but not reaching much over 103° F. (39.5° C.). With the second rise in temperature a characteristic rash appears, usually beginning in the palms and backs of the hands, and spreading rapidly to the arms, trunk and legs. The eruption is varied in character. At times it consists of small macules which gradually extend in size and coalesce until the patient resembles a scarlet-fever victim; but this is unusual. At other times there are small macules, with close-set, bright-red points, which have no tendency to spread, and separated by large areas of normal-colored skin. There may be many variations of these two types of eruption. Often the eruption is absent, or so faint that it can only be made out with the greatest difficulty. In the average outbreak the eruption is the rule.

The temperature is also subject to many variations. This is especially true in cases that have had a previous attack. As a rule, the second attack of the disease is not nearly so severe as the first attack; the third is even milder, and a fourth is very rare. In most instances the convalescence is quick and permanent, but in some individuals it may be greatly prolonged, and exhaustion from slight effort may last for many weeks.

In rare instances there may be hemorrhages from the mucosa of the nose, stomach, intestines or uterus. Pleurisy, pericarditis, endocarditis and meningitis occur at very rare intervals. The most important sequelæ are pains in the joints and muscles, and these often give great distress.

The *diagnosis* of dengue is based upon the sudden onset, the severe pain in the muscles and joints, the characteristic rash, the temperature, and the absence of the causative organism of diseases with similar symptoms. It may be mistaken for yellow fever, malaria, influenza, scarlet fever, measles, rheumatic fever, smallpox and tonsillitis. Yellow fever is readily differentiated by its slower pulse, jaundice and hematemesis. In malaria there are blood parasites. In influenza there is the absence of eruptions and the presence of the usual catarrhal symptoms. In scarlet fever there is

the complicating sore throat, with the enlarged glands of the neck. In measles, marked catarrhal symptoms, followed by the characteristic rash, dominate the clinical picture. In rheumatic fever there is the swelling in the joints, with pain on passive motion. It is very often difficult to differentiate smallpox until the eruption actually appears. Tonsillitis may be excluded by the throat symptom.

In the tropics enteric fever may be mistaken for dengue, but the continued fever and the constant headache, with the absence of the characteristic joint and muscle pains, usually makes it possible to give a correct diagnosis.

The mortality is practically nothing. In Australia it is said to cause one death in 1000. These occur in those under 5 and over 60 years of age.

There is no specific *treatment* available. Treatment should invariably be begun with small doses of calomel—say, of $\frac{1}{10}$ grain (0.006 Gm.)—and administered every half-hour until the bowels move freely. Great relief can be afforded the patient by the administration of salicylates in the form of aspirin or sodium salicylate. At times hypodermic injections of $\frac{1}{6}$-grain (0.010 Gm.) doses of morphin are necessary to relieve the pain. During the febrile period the patient can be made comfortable by sponging with ice-water. Cold drinks should be administered freely, and the usual fever diet should be prescribed. To protect the heart against strain, rest in bed is essential.

The best means of preventing infection is to exclude mosquitoes in the same manner as is done in yellow fever or malaria.

MALTA FEVER.

Mediterranean Fever, Bruce's Septicemia, Neapolitan Fever, Cyprus Fever, Undulant Fever and Septicemia Melitensis are the common synonyms used for this infection, which is a disease of long duration, characterized by febrile attacks with many remittances. It is caused by the *Micrococcus melitensis,* and usually is spread through goats' milk.

Malta fever is of very ancient lineage. Hippocrates described cases of long-continued fever with short apyrexial

intervals which lasted one hundred and twenty days, and is all probability was the disease we call Malta fever to-day. In the eighteenth and nineteenth centuries references were made by Howard, Hennen and Davy, which appear to have been Malta fever. During the Crimean War the incidence of the fever was greatly increased in Malta, but it was difficult to separate it from the typhoid fever which, in all probability, prevailed in close association with it. In 1886 it was proved to be a separate pathologic entity by Bruce,[20] who discovered in the spleen the micrococcus which bears his name. In 1887 he was able to cultivate the micro-organism on agar, and to reproduce the disease by inoculation into monkeys. In 1891 he was able to grow the germ from blood which had been aspirated from the spleen during life. In 1897 Wright and Semple[21] showed that the disease could be diagnosed by the agglutination of the micrococcus by the serum of patients. The British Admiralty, the War Office, and the Civil Government at Malta appointed a commission to investigate the disease in 1904,[22] and this commission showed that the micro-organism leaves the body mostly through the urine, and that it is capable of existing for a long period outside of the body. The commission also discovered that the milk of many goats would agglutinate the *Micrococcus melitensis,* and later they were able to isolate the germ from goats whose milk contained it. Manson reports[23] that, unless serum reactions are made with fresh blood and proved cultures, erroneous results may follow.

Prophylaxis along lines indicated by the etiology has resulted in a reduction of Malta fever among the British troops in Malta from 643 cases in 1905 to one case in 1910. In recent years more careful examinations have shown that the disease also exists to a considerable extent in Spain, Portugal and France.

The endemic areas of the disease lie along the coast and islands of the Mediterranean, the Punjab in India, and to a limited extent in Ceylon. Strong reported one case in the Philippine Islands,[24] but this was more than ten years ago, and diligent search has failed to reveal additional cases. A few isolated cases have been reported in England and the United States. It seems likely that further investigation

would show the presence of the disease in many other countries.

The disease is caused by the *Micrococcus melitensis* (Bruce). Castellani[25] expresses the opinion that a closely allied microbe is associated with the disease in addition to the specific micrococcus. The micro-organism is found in the spleen, liver, kidney, lymphatic and salivary glands. It may be found in the stomach of mosquitoes which have fed upon patients, but it has not been possible to prove that either mosquitoes, flies or fleas are concerned in the transmission. The germ is very resistant, and will live for at least eighty days in dust, and for a period of a month in either fresh or salt water. However, it has never been found naturally in dust or water, but it has been found in 10 per cent. of the goats examined in Malta. The animals are apparently quite healthy, although occasionally chronic mastitis may be noted. The disease appears to be conveyed to man by milk of infected goats, and generally disappears when milk is rendered safe. Other methods of infection, however, are suspected. It has been assumed that it may be spread through the respiratory system by inhaling dust which has been contaminated by goats' urine, and also through abrasions, wounds or cuts. The method of spread from goat to goat is not known. Exceedingly minute quantities of a culture are necessary to infect. The prick of a contaminated needle is sufficient. Infection is reported to have taken place through using a clinical thermometer in a country not known to be infected, by placing it in the mouth of a healthy subject after it had been used by a new arrival suffering with the disease. Persons between 6 and 30 years of age are most susceptible. Long residence in an endemic area is said to confer no immunity.

The micrococcus enters the system through the alimentary tract and causes septicemia. Enlargement of the spleen is a marked feature. This causes the disease at times to be mistaken for typhoid fever. Ross and Eyre[26] believe that the micrococcus may be conveyed by mosquitoes, but definite proof is lacking. Bruce[27] believes that one attack confers immunity. Other clinicians have expressed great doubt.

The spleen is enlarged and may weigh as much as 400 grams (14.1 oz.). It is soft and shows an increase of lymphoid cells. There is congestion of the liver, kidneys and other abdominal organs. The colon may be ulcerated, especially if there has been hemorrhage. The lungs are congested, and may show patches of consolidation.

Monkeys fed with infected milk require an *incubation period* of fifteen days; human beings, according to Johnstone, fourteen days. Cases occur with an apparent incubation period as low as six days. The *onset* of the disease is gradual. The patient usually continues at work, although he has a fever. The temperature rises gradually, and the clinical symptoms are much the same as in typhoid. After a few days severe pains, often thought to be rheumatic, occur in the back and limbs. Headaches become intense, and there is almost complete loss of appetite. The temperature by this time is usually three or four degrees above normal, and the pyrexia is often accompanied by slight sore throat and tenderness in the epigastric region. There may also be bronchial catarrh and congestion of the lungs. These symptoms continue for a number of weeks. The temperature remains continuously high, varying from 103° to 105° F. (39.5° to 40.5° C.), but after several weeks it may decline, and the patient feels somewhat better. In a few days a relapse occurs and the same symptoms reappear. This relapse may continue as long as the original fever, but it is usually of somewhat shorter duration. One relapse follows another, and the illness may extend over a period of many months. After that the temperature becomes undulating, with marked rise at night and fall in the morning. The patient becomes anemic and weak. The alimentary canal becomes irritated, and there is indigestion, which may be accompanied by constipation or diarrhea. Often the gums become spongy and bleed on pressure. The spleen is enlarged and painful. The patient feels miserable, and has headache and pains all over the body. Insomnia and hysterical outbreaks are not uncommon. Acute delirium is rare. The skin is usually damp. Perspiration of a peculiar disagreeable odor, profuse in character, occurs from the beginning. It is generally worse in the morning. Sudamina generally occur after the third week. Sometimes the

joints become swollen and painful, but the skin with which they are covered seldom is reddened. The hip-, shoulder-, ankle-, and knee- joints are most commonly affected. The blood shows a secondary anemia, with a loss of 20 to 40 per cent. of corpuscles. There is a marked reduction of hemoglobin and some poikilocytosis. Phagocytic power is diminished, as well as bactericidal properties. The blood findings lead Bassett-Smith[28] to the opinion that an attack does not confer immunity. One of the best diagnostic signs upon which to rely is the agglutination which takes place in dilutions of 1 in 50 within about thirty minutes. The urine is that of a typical fever case, with the exception that there is seldom albumin or granular casts. The specific germ of the disease sometimes can be found in the urine two years after an attack. The patient now becomes extremely anemic and prostrated by the repeated attacks of fever. When improvement sets in the intermissions between the febrile attacks increase until recovery takes place. The tongue clears and the remaining symptoms abate. Convalescence may begin after the twentieth day, or sometimes the fever may last nearly a year. The average period is three months. A number of varieties have been described by some authors. In the malignant type, which comes on suddenly, with a temperature of 104° to 105° F. (40° to 40.5° C.), practically all of the symptoms occur which have been described before, in a much shorter period and in an aggravated form. In the intermittent variety the onset is very slow, and the remissions are of longer duration. There is an ambulatory type, in which the patient is not aware that he is suffering with any complaint, and often gives positive blood reactions for the disease over long periods of time. There has also been a para-Malta fever described, which is attributed to the *Micrococcus paramelitensis* of Negre and Raynaud.[29] This is not a well-recognized entity.

Ulcers provoking hemorrhage in the small and large intestines are complications of real gravity, and at times extreme hyperpyrexia, pneumonia, pleuritic effusion and cardiac failure are observed. Orchitis is common.

Neurasthenia, with its long train of symptoms, is probably one of the commonest sequelæ of the disease.

The principal clinical signs upon which to base a diagnosis are the prolonged character of the fever, the profuse sweating and the joint symptoms. Bacteriology furnishes a practically positive method of recognizing the disease, and is more dependable than the attempt to make a differential clinical diagnosis. Clinically, there is great resemblance to typhoid fever during the first and second weeks. Widal's reaction or blood-cultures will distinguish typhoid. Kala-azar also resembles Malta fever, but the typical blood-parasite is a sufficient differentiating sign.

The prognosis is usually good. The mortality may be as low as 2 per cent. in some outbreaks, but at times it rises to 13 per cent.

TREATMENT.

The treatment is symptomatic, and the use of vaccines and serums has not been successful. New serums are being constantly tried, and at times some of these appear to be of value, but so far they have not stood a prolonged test. Excellent nursing is an essential. It is also desirable that the patient be kept in a screen-proof room or under a mosquito-net. Precaution should also be taken to disinfect the stools and the urine, as well as the other discharges. Open abrasions in attendants should be sealed. Headaches may be relieved by acetanilid, but at times morphin is necessary. The joint symptoms may be treated by hot fomentations or with belladonna and opium applications. The bowels should be regulated, and at the beginning of the attack fractional doses of calomel should be given until free purgation is produced. Mouth-washes of mild antiseptics should be regularly employed. Sponging to keep down the temperature is important. Briefly, the nursing and treatment should be much the same as in typhoid fever. During convalescence tonics are indicated. During fever periods the diet should be liquid.

Prophylaxis. Successful prophylaxis of Malta fever, in accordance with the experience at Gibraltar, can be insured by abstaining from the use of goats' milk, and precautions to avoid this should be taken. In endemic countries residents should be careful to use only milk that has been boiled.

CHOLERA.

Asiatic cholera is an acute infectious disease caused by the vibrio of Koch, and is characterized by violent purging, vomiting, muscular cramps, suppression of urine, husky voice and collapse.

The true origin of the name Cholera probably comes from two Hebrew words, Choli-ra (or *morbus malus*).

Among the several synonyms applied to this infection are included Cholera Asiatica, Pestilential Asphyxia, Morbus Asiaticus, Morbus Oryzeus (ascribed by Tytler to damaged rice), Haiza (Hindustani), Enerum Vandee (Tamil), Holouan (Chinese), Duba (Arabic).

Cholera is endemic in certain parts of India, and probably in China, Japan, Java and in the Philippines. It is also not unlikely that the disease is endemic in certain sections of the Balkans and the nearby Russian territory. In the past it has been assumed that the disease has advanced out of India and caused worldwide epidemics which have been responsible for a large loss of life. However, it now seems probable that the cholera carrier is an important reservoir of the disease, and that the carrier is not necessarily confined to India. The old views that cholera remained indefinitely alive in certain water supplies similar to those of the Ganges is not tenable, although it is quite probable that the vibrio may remain alive in certain water for many months. The spread of the disease is always along the line of travel. It is probably carried in the intestines of the human beings, and, perhaps, to a lesser extent in the food and water supplies which are used for human consumption. It is primarily a disease of the human intestines, and it is probably never contracted except by the introduction of the specific micro-organism into the mouth. One of the first reasonable authentic records shows that cholera advanced out of India in 1817. [There is small doubt that the disease occurred in epidemic form long before this.] Epidemic cholera has always followed the line of travel. There are, however, many instances of cholera appearing simultaneously over widely separated areas without its having been possible to trace any connection between cases. These observations, however, are confined to countries in which cholera

is endemic. Mention is made of the disease in manuscript and in ancient Chinese writings, but in view of the absence of accurate descriptions and the similarity which may exist between many of the intestinal diseases, too much reliance cannot be placed on these older writings. For instance, in London an outbreak of the disease was described by Willies in 1670 which he called dysenteria acuta epidemica. Cholera has appeared in epidemic form in recent times during five different periods (1830, 1846, 1865, 1884, 1892), and has been more or less continuously present in some parts of Europe from 1910 to 1916. In 1910 cholera was transmitted from Russia to Bari in Italy, and from there it spread through Southern Europe. In 1914 cholera spread among the Austrian troops engaged in the European war; cases have been reported even in Vienna and Prague. In 1906 and 1907 cholera was carried to Hamburg by emigrants from Russia. Probably the greatest distributors of cholera are through the pilgrimages which Mohammedans from all over the world make to Mecca. The crude conditions under which they travel, the inadequate human excrement disposal *en route* and at Mecca, favors the propagation of the disease. From Mecca it is again carried by the same pilgrims to their home countries. In 1865 cholera was carried to New York from ports in the Levant, probably *via* London, by the English ship *Atlanta,* which left London, October 10th, with a cargo of merchandise and forty persons. On October 11th the vessel reached Havre, where it remained one day, and embarked 564 new passengers, who had passed through Paris, where they had remained some days, and where cholera was prevailing in epidemic form. One day out from Havre there was a death from cholera in a child which came from the Weissen Lamm Hotel of Paris. Five other deaths followed in the succeeding five days in a family that had stayed in the hotel Huitgarderhof of Paris. On arrival of the *Atlanta* in New York, the surgeon declared 60 cases of cholera and 15 deaths during the voyage. The vessel was placed in a strict quarantine, and no cholera appeared in the United States during that year. In the early sixties cholera was imported into Quebec, and from there spread rapidly to the United States, and caused many deaths, particularly in Ohio. In September, 1892, 10

cases with 8 deaths occurred in New York, and were traced to infection imported from Hamburg; 73 cases with 43 deaths occurred in the harbor. In 1893 there were 20 cases and 4 deaths in the harbor; 1 death, which was confirmed by bacteriologic examination, occurred in Jersey City, but the disease did not gain a foothold in the United States. In 1911 almost every ship with immigrants arriving in the harbor from the Mediterranean, between the first of July and the first of September, had cases of cholera or cholera carriers aboard; 2 cases attributed to infected ships were found in Brooklyn, and 1 in Auburn, New York. More than seventeen days had elapsed in these cases since last exposure to a known case of cholera. There was, however, much evidence that there were many cholera carriers on board the vessels from which the Brooklyn cases came. During August, 1911, 2 cases of cholera occurred in Boston, 1 in an Irish-American who had been a permanent resident of Boston, and no contact with cholera cases could be established. The other case occurred in an Italian woman who had been a permanent resident of Boston for a number of months, and had not been exposed to cholera. The diagnosis in the case of the Irish-American was bacteriologically confirmed by the city health department and the bacteriologic laboratory of the United States Public Health Service. It is quite likely that cholera is transmitted from place to place and from one country to another, principally by the means of the human intestinal discharges finding their way directly or indirectly through food or water to the mouths of other individuals.

A severe outbreak of cholera began in the Philippines during March, 1902, and continued until April, 1904, during which time 166,252 cases and 109,461 deaths occurred. The origin of this epidemic has been attributed to an importation of infected cabbages from Hong Kong. It is more than likely, however, in view of the present knowledge, that in spite of the rigid five-day quarantine which was imposed against Hong Kong, where cholera had prevailed for some weeks previous to its appearance in the Philippines, the disease was probably introduced by a cholera carrier. It is also of interest to note that the disease made its appearance simultaneously in Manila and in Nueva Caceres, which is three

days' steaming distance from Manila. In August, 1905, the disease again reappeared and lasted until April, 1907, during which time 13,429 cases and 10,093 deaths occurred. It again appeared in July, 1907, after an absence of only two months, being present until March, 1911, with a total of 50,871 cases and 33,792 deaths; was absent from April through June, 1911; and again present from July through October, 1911, with 50 cases and 43 deaths. From November, 1911, through July, 1913 (a year and nine months), no cases of cholera were reported in the islands, but in August, 1913, the disease again appeared, lasting through March, 1914, with a total of 1093 cases and 780 deaths; from April through June, 1914, being responsible to date (January 26, 1915) for 3004 cases and 2041 deaths.

There is considerable evidence to show that the disease prevails in similar epidemic form in Java, the Straits Settlements, Japan, and, to a more limited extent, in China. The small number of cases in China is, in all probability, due to the great value of human excrement as an insecticide and fertilizer, which makes its immediate collection of great commercial advantage; and also to the fact that the Chinese do not eat with their fingers, and almost invariably drink boiled water, in the form of tea, and eat almost all of their vegetables in a cooked state. There is considerable reason to believe that, on account of the other filthy habits of the Chinese, if they did not have the foregoing customs, the ravages of cholera in that country would be frightful. In Japan the same value is placed upon human excrement as in China, but the Japanese drink much unboiled water and eat many contaminated vegetables in a raw state.

It is likely that cholera microbes may be transported in water or foodstuffs which act as a suitable media. That cholera may be transported by healthy individuals who act as carriers has now been proved. During the cholera outbreak in Manila, in 1911, McLaughlin showed that 5 per cent. of the prisoners of Bilibid were cholera carriers, although none of them had been exposed to the disease. In August, 1916, there were over 150 cholera carriers in Bilibid Prison, but no true case of cholera occurred. The diagnosis in each instance was bacteriologically confirmed by Schobel, of the

Bureau of Science Laboratory. Cholera carriers were detected in Bilibid Prison for a number of months prior to August, which corresponds to the period in which cases of cholera were constantly occurring among the residents of Manila. So far as known, there was no contact of these prisoners with infected cases outside of the prison.

Cholera also may be transmitted by animals. In 1914, Barber,[30] of the Bureau of Science at Manila, showed that cockroaches which fed on human cholera feces may harbor cholera vibrios in their intestines, and that they may occur in enormous numbers in the insects' feces for at least two days after the last feeding. It is, therefore, apparent that, by means of feces and vomited matter, cockroaches may act as carriers of cholera to human food. Barber also showed that cholera vibrios from cockroaches' feces would survive on a pie, for instance, for at least sixteen hours after being deposited.

Also that cholera vibrios in human feces, when placed on food in competition with other bacteria, will survive for at least four days. He also found cholera vibrios in the bodies of ants at least eight hours after they had ingested cholera cultures which had been placed on human feces. The more probable routes by which cholera germs find their way from infected human intestines to the gastro-intestinal tract of other individuals probably are as follows: In widespread epidemics probably through the infection reaching the water supplies, and in more restricted outbreaks probably through the micro-organism reaching the food by the means of infected hands, or by flies, or other insects. There is an authentic case on record in which flies transmitted the disease to the nursing bottle of an infant. Cultures have been made from the feet of flies which had come in contact with infected human excrement. There are instances on record where whole rivers have become infected, and the disease spread down these rivers to people who used the water. It should also be remembered that there are instances in which the disease has spread up rivers. This is explained by cholera carriers. The theories of Pettenkofer, that the vibrio is propagated in the soil, and that an outbreak of the disease is intimately connected with the rise and fall of subsoil water,

are not tenable, and lack adequate data for their clear proof. Cholera is mostly a disease of warm countries, although very severe outbreaks have occurred in countries of the temperate zones, but this usually took place in the summer time, when the conditions were tropical. It is more than likely that the reason why the disease does not spread during the winter period in the temperate zones is the fact that cold weather is inimical to the growth of cholera vibrios in the human feces after they have left the body, and there is, in consequence, less opportunity for the infection to be spread by insects or hands.

Strictly speaking, cholera cannot be said to be a race disease, although in Manila, for instance, the statistics show that one Chinaman out of 7000 contracted the disease, whereas one Filipino out of 300 became infected. This is doubtless due to the personal habits of the two races; a Chinaman, for instance, practically never drinks water except in the form of boiled tea, and uses cooked food which he eats with chopsticks. A Filipino drinks unboiled water, and eats his food, much of it uncooked, with his fingers, thereby affording an excellent opportunity for a cholera carrier to transmit the infection to the common bowl which serves so frequently as a receptacle from which two or more persons dip with their fingers to partake of their food.

Cholera is caused by the vibrio of Koch, although the presence of this micro-organism in the human intestines does not necessarily mean that the individual has or has had cholera. It is has now been well demonstrated that many persons, especially in districts in which cholera prevails, harbor cholera vibrios, but do not have the disease. This fact has become so generally recognized that literature of the present day fairly teems with the question of cholera carriers. There are instances on record in which organisms identical with the cholera vibrios have been found in the stools of persons who are residents of the Mississippi Valley in the United States. In some of these areas it can be said with reasonable certainty that no cholera has occurred for more than twenty years. Another interesting phase of the question concerns the sudden appearance of cholera in communities, without its being possible to trace the infection to known centers of

cholera. This fact has frequently been observed in Manila, and very extensive investigations have been made to trace the infection to an outside source, but without success. In 1914 it was thought desirable in Manila to examine for cholera vibrios the stools of persons who had been discharged as cured from the cholera hospital the year previous. In the first fifty examinations made, an active cholera carrier was found. He was employed as a waiter in a restaurant, and his record showed that he had been discharged from the cholera hospital seven months previously, and only after two negative stool examinations had been reported. About a month after the discovery of this case cholera suddenly reappeared in Manila. There have been also outbreaks of cholera in Manila which were preceded by deaths suspicious of cholera, but in which it was impossible to demonstrate the cholera vibrio. These cases usually died with an intense nephritis, evidently of toxic origin, the exact source of which could not be traced.

Some authors have attempted to explain outbreaks of cholera on the basis that cholera vibrios are more or less normally present in the human intestines, and that mental worry and other depressing factors may so reduce the vitality of the subject that the germs are able to overcome the defenses of nature and cholera results. An explanation of that kind is unsatisfactory. An explanation that is more sound, although unproved, is that there is a change in the morphology of the micro-organism, and that during certain cycles the vibrio is non-infective. Also that the micro-organism becomes so attenuated as to be non-infective, and that it is only by the passage through one or more human intestines that it attains sufficient virulence to be infective; thus conditions arise that are then favorable for an outbreak of cholera.

Cholera is directly transmissible from man to man. An example of this form of transmission is well illustrated by doctor or nurse whose hands come in contact with the discharges of a cholera case, and who then partakes of food without disinfecting the hands, the penalty of this gross delinquency being a typical attack of the disease. Great epidemics are probably caused by the water supply becoming infected with cholera-infected human excrement. A classic

instance of water infection is that of the London Pump, in 1854, when the well became infected, and persons who used the water contracted the disease. In 1892 the cholera outbreak in Hamburg was confined to persons who used the unfiltered water of the Elbe. Those who used the filtered water escaped. The micro-organism was found in the river and in the tap-water. In 1911, at Cebu, a real "nest" of cholera was traced to an infected well; several Americans and a great number of Filipinos who used water from the well lost their lives. Milk is frequently an important source of infection by being diluted with cholera-infected water, and, once contaminated, it makes an excellent media for the growth of cholera vibrios. Great epidemics are caused by extensive water or food infection. The hands of persons who come in contact with cholera stools and then handle food are a great factor in the dissemination of cholera. The human cholera carrier is also a very important factor, especially since he introduces the vibrio into many places in which its presence is not suspected. It must also be remembered that cholera germs can live in water under favorable conditions for more than one hundred days, and that they have been reported to have remained alive in human feces for a period of one hundred and sixty-three days. Cholera always follows the routes of travel, whether by human, ships, rivers, roads, railways, or other means of communication. It is also of importance to draw attention to the fact that spirilla resembling cholera vibrios are frequently found in human discharges, and that it is only by careful cultivation and agglutination tests that the cholera vibrio can be definitely identified.

Until quite recently it was believed that the cholera vibrios could be found only in the intestine, and that the symptoms and the effects of the disease were due to the production of an endocellular toxin. Grieg[31] has lately demonstrated the presence of the cholera vibrio in the spleen, but up to the present time it has not been possible to state whether the vibrios outside of the human intestines are connected with the symptomatology.

In general, however, it may be stated that the vibrio occurs abundantly in the glands and the epithelial cells and mucosa of the small intestine. It is presumed that an endo-

toxin which is set free causes the gastro-intestinal disturbance which is responsible for the passage of fluid from the blood into the bowel. Analyses show this at first to consist of water, then sodium chlorid and other inorganic salts, still later phosphate and potassium salts, and finally organic substances. The fact that water is found first would indicate that the process is not an endosmosis. It may be a secretion. At all events, it causes great concentration of the blood, the specific gravity of which may reach to 1.078. In this concentrated form sometimes as many as 8,000,000 erythrocytes per cubic millimeter are found. There is a marked fall in the blood-pressure, which may decline to as low as 50 millimeters of mercury (systolic). The urine is generally suppressed or scanty, with high specific gravity, albumin, casts, and an increase in the amount of indican.

As a rule, the *post-mortem* rigidity is marked. It sometimes happens that bodies dead of cholera change position; sometimes they rise to the sitting posture, or are even thrown from the table by severe muscular contractions. Occasionally contractions which force air out of the lungs produce sounds which strike terror into the hearts of those who are not familiar with the cause of their production.

On cutting the tissues it is noticed that they are very dry, and that the blood is frequently thick and tarry in appearance. On opening the peritoneal cavity a sticky sensation is imparted to the hand when it is passed among the loops of the intestines. This is usually very characteristic, and is peculiar to cholera. The stomach nearly always contains fluid, and when death has been sudden it may contain food. The intestines are reddish in appearance, and there are often small punctiform hemorrhages. The reddish appearance, however, is not specially characteristic, and is much the same as seen in other conditions. The contents of the intestines are usually found to be a whitish turbid liquid, very much like the sauce commonly used for cottage pudding, but somewhat thinner. Often it contains small whitish flakes, and on microscopic examination is found to consist of food particles, epithelial cells, red and white blood-corpuscles, mucus and micro-organisms. The mucosa of the stomach and intestines is generally hyperemic and swollen, and may be marked with

numerous punctiform hemorrhages. The villi are swollen, giving the surface a dull, opaque, translucent appearance, and Peyer's patches and the solitary follicles are much enlarged. The ilium and the upper end of the large intestine appear to be studded with cooked sago-like grains. In the lower bowel there are usually large quantities of the turbid whitish fluid described above, but if death has been due to cholera sicca the stools are usually hard and firm, and cholera flakes are rare. The glands of the stomach and duodenum are enlarged, and the surface is denuded of epithelium. There are no special lesions of other organs. The liver is seldom enlarged, and only rarely does it show moderate cloudy swelling. The spleen is usually normal, but may be small, hard, and wrinkled upon its surface, and deep red upon section. The kidneys have a characteristic deep-red and opaque appearance, this change being most marked in cases in which anuria was present some days before death. The heart is usually soft and flabby, and the muscles degenerated. In cases in which death has been rapid there are frequently ecchymoses on the surface of the endo- and peri- cardium. In cases which have been ill over three days there may be evidences of cholera pneumonia. Microscopically, the cholera vibrios may be seen in Lieberkuhn's follicles, in the epithelial cells, and in the mucosa of the intestine and the stomach. Rebulski and Grieg[32] have recorded cases in which the vibrio was found in the liver, spleen and heart.

Spirillum of Cholera. The cholera vibrio was discovered by Koch[33] in 1883 in Egypt, and he confirmed his work the following year in India. In the intestines and dejecta the organism presents the appearance of a short thick rod slightly curved in the long diameter. It is about half as long and twice as thick as the tubercle bacillus. The cholera vibrio is 0.8 to 3 microns in length, and 0.3 to 0.5 microns in breadth. It is generally found singly, but it may be arranged in pairs with the curves opposite to each other, in which event it may resemble the letter S. A simple microscopic examination of a smear preparation is not sufficient to establish or to exclude a diagnosis. It very often happens that curved spirilla cannot be found in smear preparations, but nevertheless they may be recovered by culture. The motility is very charac-

teristic, and has been demonstrated by Löffler to be due to fine celia which are longer than the bacteria, and are attached to one end of the micro-organism. Many different methods for the bacteriologic diagnosis of cholera have been devised. The method used by Schöbel and other workers, used many thousand times at the Bureau of Science in Manila, has proved reliable, quick and practical:

"The stool specimen, which is collected with a sterile glass tube, is placed into a tube containing a slightly alkaline 1 per cent. peptone solution. This is incubated for some twelve to twenty hours, and then transplanted to Dieudonnes plates. On these plates practically no other organisms are found except those of cholera, and the growth of the cholera organisms is characteristic. A small amount of culture is then taken from Dieudonnes plate and brought in contact with the cholera serum, and the coagulation may easily be observed by the naked eye, with the characteristic clumping under the microscope. For making examinations of cases of cholera which occur in places in which laboratory methods are not available, it has been found that if stool specimens taken with sterile glass tubes are placed into agar tubes the organism may be recovered from such transplants up to five days from the time that the specimen was taken. These tubes are kept at ordinary temperatures in the Philippines, which usually range from 26.7° to 35° C. (80° to 95° F.). These tubes are placed into the agar cultures and mailed in ordinary mailing tubes.

"Schöbel[34] in 1914, in Manila, found that the cholera organisms live for six days in distilled water, thirty-three days in ordinary Manila tap-water, and over one hundred and six days in salt water. Certain food may be of great importance in transmitting the organism, as in the case of milk, in which it grows without producing any visible alteration. It has been shown that the organism would live for at least two days in milk and forty-eight days in butter. According to Koch a solution of 1 to 400 of carbolic acid and 1 to 25 of sulphate of copper and 1 to 10,000 of corrosive sublimate are sufficient to arrest development. Ashburton[35] has shown that creolin, 1 to 2000, destroys the vibrio. The cholera organism has but feeble resistance to drying and to sunlight; it is easily

killed with a weak germicidal solution. In general, it may be stated that the organism will not remain alive in the ordinary drinking-water for more than six to seven days."

Cholera is generally said to have an *incubation period* of from two to five days, but in actual practice it is found that the incubation period is seldom more than forty-eight hours. The cholera conference at Constantinople concluded as follows: "That all the facts cited in regard to a period of incubation longer than a few days are based upon cases that are not conclusive, either because the premonitory diarrhea was comprised in the period of incubation, or because the infection could have occurred after the departure from the infected locality."

It quite frequently happens that the appearance of the first *cholera symptoms* is preceded by prodromata in the form of diarrhea or a feeling of malaise. The onset is usually sudden. The attack begins with diarrhea, usually attended by colicky pains in the abdomen. Vomiting generally occurs later. At first food is vomited, and this is followed by watery fluid with bile and occasionally blood. The motions at first are formed in character, but soon assume the so-called rice-water appearance, with numerous white flakes which, when examined, are found to consist of mucus, which contains vibrios and epithelial cells. Sometimes blood is passed, but this is rare, and frequently will be found to be due to hemorrhoids or to some other local cause. The thirst usually becomes severe. As the purging and vomiting continue, the urine diminishes, and generally stops altogether. The blood leaves the subcutaneous tissues, which causes contractions, the facies alter, the nose becomes sharp, the cheek-bones prominent, the eyes sunken, and the skin of the fingers becomes wrinkled. Similar wrinkling takes place about the feet. The circulation is profoundly affected; the systolic blood-pressure falls, often to as low as from 20 to 60. The pulse becomes quick and rapid, the heart-sounds enfeebled, the lips pale, the nails bluish, respiration rapid, the voice characteristically husky, and severe cramps appear in the muscles, especially in the arms and legs. The mind is generally clear, but the patient is apathetic except during the agony produced by the painful cramps. The skin feels cool and clammy, and the axillary temperature falls

below normal, although the rectal temperature may be above normal. The patient quickly passes into the second stage of the illness and, unless improvement takes place, the so-called algid stage comes on. The pulse disappears at the wrist, the heart-sounds become weak and irregular, and the urine completely suppressed; the diarrhea ceases, and the patient becomes comatose, death supervening within from twelve to thirty-six hours after the onset of the attack. If the patient is to recover the diarrhea diminishes, the skin becomes warmer, the pulse and blood-pressure improve, the kidneys secrete, the subcutaneous tissues gradually absorb a normal quantity of fluid, and the appearance of the subject gradually returns to normal. Probably the most favorable symptom is the re-establishment of the renal secretion. Cases sometimes recover in which there has been a complete suppression of urine for more than seventy-two hours. Cholera is very often spoken of as occurring in three stages. First, the stage of invasion, during which there is free evacuation; second, the stage of urinary suppression; third, the stage of reaction or death. In brief, the prominent symptoms may be summed up as subnormal temperature in the axilla, husky voice, severe cramps in the legs and arms, suppression of urine, whitish turbid fluid stools, absence of fluid in the tissues, and cool, clammy, profusely perspiring skin and thready pulse. These make a combination so striking that it is never forgotten for those who have seen the disease. The temperature often falls to 35° C. (95° F.) in the mouth. Cholera is frequently spoken of as occurring in the following varieties:

1. *Ambulant cases,* usually now referred to as cholera carriers, who show no symptoms of the disease except the presence of the cholera vibrios in the stools.

2. *Choleraic diarrhea,* characterized by severe purgation and the passage of yellow stools which contain cholera vibrios. Recovery may take place before the patient passes into a typical attack of the disease.

3. *Cholerine,* characterized by active abdominal pains, numerous feculent stools, followed by rice-water diarrhea lasting for about a day, with recovery of the patient without further symptoms. True cholera vibrios are not found in these cases.

4. *Cholera sicca,* a fatal type of the disease in which death occurs before the typical symptoms of diarrhea and vomiting appear. Bacteriologic examination of the stools shows cholera vibrios almost in pure culture. In this type of cholera it sometimes happens that persons are stricken while walking on the street, and, suddenly falling to the ground, die before assistance can reach them. At other times the victim is seized during the night, and found dead in bed in the morning.

The most common complication of cholera is pneumonia. Nephritis is often given as a complication, but this must be regarded as one of the most constant lesions of cholera. At times gangrene of the lungs may occur. Abortion in pregnant women is the rule. Secondary affections, however, are very common. A fever resembling typhoid sometimes occurs, but it is always of shorter duration than true typhoid. Various skin eruptions may occur. As is to be expected, a severe illness like that of cholera is frequently followed by permanent damage to the health of the individual. Extreme anemia and a tendency to diarrhea and digestive disturbances may persist for a long time after convalescence from the active choleraic clinical picture.

The *diagnosis* of cholera during the presence of an epidemic is very simple, but the first few cases of an outbreak are frequently overlooked. The following symptoms, however, should always lead to a careful bacteriologic examination of the stool, in order to ascertain whether cholera vibrios are present. Cases of sudden illness in which there is vomiting and purging, subnormal temperature, suppression of urine, clammy perspiration, husky voice, severe cramps in the arms and legs (especially in the calves), weak thready pulse, and wrinkling of the skin of the hands and feet are tentatively to be labeled cholera.

The *prognosis* of cholera is unfavorable in the general run of cases. At the beginning of an outbreak the mortality is often as high as 90 per cent., whereas toward the end of an epidemic the death-rate often declines to about 15 per cent. A fair average mortality for a complete outbreak would be about 50 per cent. Probably the large amount of conflicting evidence as to the value of different treatments for cholera

may be explained on the grounds that the remedies were given at different periods of the epidemic; a treatment that apparently at the beginning of the outbreak was of no great value might, when used by another observer toward the close of an outbreak, apparently give good results.

TREATMENT.

The great number of remedies advocated for cholera are probably the best evidence that no satisfactory treatment has been found. On an experience based upon many thousands of cases, it may be stated that the following probably produces as satisfactory results as any other. The patient is placed in a warm bed with artificial heat, supplied by hot blankets, by hot-water bottles, or by electric pads, the object being to restore the axillary temperature to normal. A preliminary dose of ½ grain (0.03 Gm.) of calomel often seems of service. Fluid by mouth is given freely. Vomiting is a more or less constant symptom, and the use of water, which is of value in cleansing the stomach, does not seem to increase it. At least a certain amount is absorbed, and perhaps aids in the elimination of toxins. Intravenous injection of normal salt solution is employed at intervals of a few hours, and in quantities designed to produce a full pulse, the proper quantity required being approximately from 1 to 2 liters (quarts). In the course of twenty-four hours a total volume of 5 liters (quarts) may be necessary. A stimulant like strychnin in $\frac{1}{60}$-grain (0.001 Gm.) doses is usually necessary. Morphin is given in sufficient amounts to make the patient comfortable. The treatment advocated by Rogers[36] with strong alkaline salt solution has not given any better results at the San Lazaro Hospital in Manila than ordinary salt solution. In brief, any measures which will induce the skin to take over the work of the kidneys should be employed. In practical experience it has been found that in all cases in which there is no marked suppression of the urine the patient will generally recover without any treatment whatsoever.

Prevention. Two principles govern the measures which should be taken to avoid cholera. The first looks to the exclusion of living cholera spirilla from the gastro-intestinal

tract, and the second refers to preserving any natural resistance, particularly on the part of the intestines, to injury due to the presence of that organism in the intestinal contents. It is believed that the toxins produced by the cholera spirillum are not absorbed through intact intestinal epithelium, but gain entrance to the circulation only after the epithelium has become damaged, necrosed, or desquamated. Therefore all dietary indiscretions which might cause intestinal irritation are to be avoided. Overfatigue and exposure to cold are to be carefully guarded against, and it is advisable not suddenly to change the ordinary mode of life, but simply to follow the usual habits in a temperate manner. The free use of alcohol appears to be extremely dangerous; those addicted to its use are particularly susceptible, and when attacked by the disease the prognosis is bad.

Care in the character of the food is important. All articles which might carry infection should be avoided entirely, or partaken only after being cooked. This is especially the case with water and milk, which should be heated to the boiling point. Prolonged boiling is not necessary, but to insure the death of the spirillum it is safest actually to boil liquid articles of food. As a prophylactic measure it has been recommended to take acid drinks, such as very weak hydrochloric acid, but it seems doubtful whether this is a wise measure, in view of the danger to the digestion if sufficient acid be taken to act as an effective agent in killing the spirillum. Furthermore, there is also the objection that the use over a period of several days of these acidulated solutions produces serious digestive disturbances, and that during such period there is interference with the hydrochloric acid and other gastric secretions, and, therefore, a greater susceptibility to cholera than if such solutions had not been taken. Particular care should be exercised against infection by those attending cases of cholera. They should disinfect the hands immediately after they have come in contact with the dejecta or vomitus from the patient. A 2 or 3 per cent. solution of carbolic acid, or 1 : 1000 solution of bichlorid of mercury may be used for this purpose. Chlorid of lime solutions are not desirable, owing to the fact that no dependability can be placed upon the amount of chlorin which they may contain.

Success in preventing the spread of cholera depends solely upon the destruction of the spirilla, and this means the safeguarding of the entire human excreta of any community in which cholera appears. A campaign against cholera resolves itself into disinfecting or otherwise safeguarding human excrement. In North American cities this is largely safeguarded by the water carriage of sewage which prevails almost everywhere, so that in such communities the outbreak of a large cholera epidemic is almost impossible. The next important step is to safeguard the water supply. Even in the case of water supplies that are derived from uninhabited watersheds, it is believed now to be strictly indicated that all such supplies should be safeguarded by the introduction of calcium hypochlorite in quantities which have been determined by an expert to be suitable for the water to be treated. Cases of cholera should be promptly isolated and placed under the care of competent persons, so that the stools and vomitus may be promptly disinfected, as well as all materials with which they may have come in contact. For this purpose 5 per cent. carbolic solution used in two or three times the bulk of the material to be disinfected perhaps is still the most reliable and efficacious disinfectant, although many of the coal-tar derivatives may be used if carbolic acid is not available. All contacts of cholera cases should be promptly examined, in order to determine whether they are cholera-vibrio carriers, and, if so, to be isolated like actual cholera cases. Places in which cholera cases are kept should be strictly safeguarded against flies, because there is great danger of disseminating the disease from this source. Another common means of spread is by the hands of individuals who have assisted persons who have been stricken with cholera, and who do not realize that they may have become infected. In more primitive communities it has frequently been found possible, after other means had failed, to eradicate cholera by requiring the entire community to immerse its hands in a bichlorid solution. Markets where food articles, like meat, fish and vegetables, are sold and are handled with the hands, have often been safeguarded by requiring every person who entered the market to place his hands in a barrel of bichlorid solution conveniently kept at the entrance of the market. In slum sec-

tions in which cholera appears it is important to make stool examinations for the cholera vibrio of all members of such crowded communities. Maritime quarantine should be restricted to the stool examinations of persons who have been in cholera districts or who have come in contact with such persons. It is no longer necessary to place quarantine upon merchandise, because many practical experiments have shown that, even in articles which are usually contaminated, it is impossible for the cholera organism to live for more than five days, except in a water supply. The water supply of ships coming from cholera-infected communities should always be disinfected with permanganate of potassium. In communities where latrines are commonly used, a pail system should be immediately installed. If this is not practicable, the whole latrine should be disinfected and closed, and temporary pits dug, which can be covered over with lime or fresh earth immediately after their use. The dead should be wrapped in bichlorid sheets and placed in hermetically sealed coffins to guard against the discharges leaking therefrom before the body finally reaches the grave or is cremated. In brief, the measures to apply in any given community in which cholera appears will readily suggest themselves to any sanitarian when he remembers that the whole question resolves itself into the prevention of human excrement from cholera cases or carriers reaching the mouth of human beings, and that, in order to be certain that this does not happen, the human excrement of the entire community must be safeguarded along the lines already indicated.

BERIBERI.

As synonyms for beriberi the following are current: Polyneuritis Endemica, Neuritis Multiplex Endemica, Hydrops Asthmaticus, Synclonus Beriberia, Myelopathia Tropica Scorbutica, Paraplegia Mephitica, Serophthisis Perniciosa Endemica, Panneuritis Endemica, Berbiers, Kakke (signifying a disease of the legs in Japan and China), Loempoe (Java), Kaki-lem-but, Hinchazon de los Negros y Chinos, Maladie des Sucreries (French Antilles), Hinchazon (Cuba), Inchacáo, or Pernerias (Brazil).

Beriberi is a serious disease, the effects of which manifest themselves principally by degenerative changes in the nerves, by heart attacks, by dropsy, and frequently by making a cripple of the person attacked.

As beriberi is intimately connected with the effects of nutritional disturbances, it is more than likely that the disease has been present from the very earliest times. According to Scheube,[37] beriberi is mentioned by Strabo and Dion Cassius as having attacked the Roman army while in Arabia in 24 B. C. Kakke is frequently mentioned in early Chinese writings, and it is minutely described in a pamphlet of the seventh century.



Fig. 9.—A healthy chicken, used in beriberi research.

It is also recorded as having occurred in Japan in the ninth century. Bontius of Europe described the disease under the term beriberi in 1758. Tulpius, a Dutch physician, described the disease in a person who had returned to Holland from the East Indies prior to 1800. Since then the literature of the disease has steadily grown. There is much reason to believe, however, that a number of other diseases have been confused with beriberi. It is quite probable that uncinariasis has been frequently mistaken for it. There is much reason to believe, however, that the incidence of the disease in the Orient has markedly increased since the advent of steam rice-mills. The old hand process of husking and cleaning the rice did not, as a rule, remove the cortical layer of the grain.

There have been notably large numbers of deaths due to beriberi in public institutions, in the Japanese Navy, on ships,

jails, insane hospitals, and other places where polished rice has been the staple article of diet. In 1904 and 1905 it is stated that 24 per cent. of the entire sick and wounded in the Japanese armies were disabled by it. It is estimated that there are annually at least a hundred thousand deaths and a half-million cases of illness due to beriberi throughout the Orient. There is much reason to believe that the disease in some countries is indirectly associated with infant mortality and morbidity. In Manila, for instance, where over one-half of the children die before they reach their first birthday, the mortality is much greater among breast-fed children than



Fig. 10—Same chicken. Paralysis after being fed exclusively on white rice for four weeks.

among bottle-fed children. The health of breast-fed infants of mothers who subsist on polished rice as a staple article of diet improves in a remarkable manner when the extract of rice polishings is fed to such infants.

Beriberi prevails extensively in Japan, China, Philippine Islands, Borneo, Indo-China, Straits Settlements, Federated Malay States, Java, and Sumatra in the Eastern Hemisphere, and in other places where people whose staple article of diet is polished rice have migrated. It is particularly common on vessels which have Asiatic crews. In the Western Hemisphere the disease has been frequently reported in Brazil, also among the natives of Iceland, and in these countries is due to an unbalanced ration.

That beriberi is intimately associated with diet cannot longer be successfully disputed, although there are still a

number of observers in various parts of the world who cling tenaciously to the infection theory, or maintain that the cause of the disease has not yet been adequately demonstrated. Without concerning ourselves at great length with the various theories which have been advocated, it may be said without successful contradiction that beriberi can be completely eradicated among a people whenever a properly balanced diet is used by them. The practical evidence in support of this contention is overwhelming. In the Philippine Islands, for instance, where more than a thousand deaths occurred annually in public institutions, as in the leper colony, insane hospitals, prisons and orphan asylums, the disease immediately disappeared when an unpolished rice diet was substituted for the polished rice, and reappeared when polished rice was again used. Convincing proof that polished rice was an insufficient diet became unexpectedly available in the Culion Leper Colony when the disease reappeared in 1912. An investigation showed that during the autumn of 1911 it became impracticable for the Philippine Government to obtain an adequate supply of unpolished rice, and in November, 1911, the use of polished rice was begun at Culion and continued until February, when unpolished rice was substituted and has been used ever since. In January, 1912, there were 2 deaths from beriberi; in February, 36; in March, 30; in April, 3; and since that time there has been no further record of deaths from beriberi in Culion, or in any other Philippine Civil Government institutions. This experience shows that in a population involving approximately three thousand people, among whom beriberi was continuously present for more than three years, the disease disappeared when unpolished rice was used, reappeared when polished rice was substituted, and disappeared again when unpolished rice was used. Similar experiments have been reported by Fraser and Stanton[38] in the Federated Malay States; by Highet[39] in Siam; by Schüffner[40] in Sumatra; Eijkmann[41] in Java, and Shiga[42] in Japan. Acting on this knowledge the principal civil medical officers—Ellis in the Straits Settlements, Sansom in the Federated Malay States, Cobb in Borneo, and Highet in Siam—have been encouraging the use of unpolished rice in civil institutions, and the number of cases of beriberi have fallen in

direct proportion to the completeness with which the unpolished rice was substituted for the polished rice.

There has been considerable difference of opinion as to what constitutes the essential lesion of beriberi. It has frequently been referred to as a disease of the blood, by others as a disease of the arteries. Since the extensive work of Scheube and of Baelz,[43] beriberi has been considered to be a peripheral neuritis. There is, however, much evidence to show that the disease is of central origin, and that the spinal cord and brain are involved. As beriberi is a nutritional disease, and as the symptoms are due to faulty metabolism, it seems logical to assume that before the peripheral nerves are attacked there must be disturbance of the central nervous system.

On inspection in cases of so-called wet beriberi, the whole body is swollen, especially the lower extremities. On opening the abdomen large quantities of fluid are found. There is also an excess of pericardial fluid. There is nothing specially characteristic about any of the abdominal organs that would not be found in cases of dropsy due to other causes. The heart is generally hypertrophied. There is serous effusion into the pericardium, pleural cavities, peritoneum and cellular tissue. This tendency to effusion with cardiac dilatation aids to distinguish beriberi from other forms of multiple neuritis. A lesion of the duodenum which is referred to as a duodenitis is reported to be present by many authors, but it is not a characteristic lesion, and its presence is actually disputed by other authors. Microscopically, the infected nerves on section show the characteristic lesion of nerve degeneration.

The vagi probably show the most constant nerve lesions, and there are changes in the vagal nucleus and the vagal ganglia. The peripheral nerves of the limbs are next most frequently affected.

In dry beriberi, with the exception of the lack of effusion, the *post-mortem* findings are almost identical with those of wet beriberi.

In acute cases the right heart is always dilated, and is hypertrophied in older cases. The left ventricle may be moderately dilated, but it is rare to find it hypertrophied. The

myocardium generally shows fatty degeneration and round-cell infiltration under the endo- and epi- cardium. These heart changes, according to Scheube,[44] are the same as found in rabbits when both vagi are cut.

The muscles contain many atrophied fibers among the normal. The diseased fibers first lose their striation. Colloid degeneration takes place, with proliferation of the nuclei of the sarcolemma. As the atrophy proceeds, the connective tissue increases.

It is usually stated that the *incubation period* of beriberi is unknown. Judging from an experience based on observation of several thousand persons, it is probable that the incubation period, where polished rice and fish are the staple articles of diet, is about sixty days. Beriberi may be described as occurring in three forms. First, dropsical beriberi; second, dry beriberi; third, low-grade beriberi. The disease may be acute in onset. Although the great majority of cases begin very insidiously, the patient usually loses appetite, often has pain in the abdomen, and frequently complains of nausea. Incipient beriberi can often be detected by the pain provoked by making pressure over the epigastrium. The temperature is usually normal. Probably one of the first definite clinical symptoms of the disease is associated with the heart, manifesting itself by a sensation of oppression over the pericardium. There is visible throbbing of the vessels of the neck, epigastric pulsation, cardiac palpitation, dyspnea, and marked acceleration of the heart upon the slightest exertion. The right side of the heart is dilated, and a hemic murmur may be heard. There is a decrease in urine which is attributed to the cardiac insufficiency, and in the wet form dropsy appears. This may be confined to slight edema over the tibia, or may be very extensive and consist of effusions into the peritoneal, pericardial and pleural cavities.

In the beginning of the attack there are exaggerated knee-jerks and a sense of heaviness and lack of control in the legs. Gradually the knee-jerks diminish, and finally disappear altogether. The patient may become incapable of walking, but before reaching this stage acquires a very peculiar gait, which somewhat resembles that of locomotor ataxia. He usually walks with the legs wide apart, cannot stand with closed eyes,

and has a sensation of walking on something soft. The calves become tender, and various forms of hyperesthesia appear, especially about the legs and arms. The anterior tibial and peroneal muscles seem to be more tender and waste faster than other muscles. The forearms may be paralyzed, with wrist-drop and loss of power in the grip. The muscles gradually waste, and electrical reactions of degeneration set in. In the early stage of the disease a very rough physical test for beriberi is the inability of patients to jump on a box 12 inches (30.4 cm.) high. The paralysis spreads to the muscles of the calf, thigh, gluteal region, arm, hand, and, in severe cases, to the muscles of respiration, and, in rare instances, to the ocular muscles. Various paresthesias occur. The patient often has the sensation of touching things with gloves on. The sensations of heat, cold and pain are often lost. This usually begins in the feet and spreads upward. The numbness of the fingers prevents the patient from performing many simple acts, as, for instance, buttoning the collar, sewing, and working with tools. The innervation of the heart is much affected, and gives rise to many indefinite murmurs. Palpitation and epigastric pulsation are very common. The pulse is usually increased in frequency, and is of low tension. Death usually results from cardiac failure. The blood does not show much abnormality, although there may be some evidences of anemia. The urine is usually diminished when there is edema, but the volume greatly increases when the patient begins to improve.

There is no great clinical difference between the wet and dry forms. On inspection, however, the distinction between the two forms of the disease is most striking. One has the typical appearance of dropsy, and the other shows great emaciation. According to some authors, the wet and dry forms of the disease are only the early and late stages of beriberi. This, however, is disputed. Some authors state that the difference in the two forms of the disease is actually due to differences in the action caused by substances or the absence of substances in the food. The digestion is usually fair, the tongue clean. Vomiting is regarded as an unfavorable symptom. Constipation is frequent, and the temperature may be normal, or even subnormal. The quantity of urine depends largely on the stage of the disease; when dropsy is

passing off there is an increase in the amount of urine. Sometimes the larynx is partly or completely paralyzed, and the voice is raucous, or even completely lost. Death may occur during any stage of the disease, and frequently follows any unusual strain that may be put upon the heart, as, for instance, attempting to rise in bed, or walk or climb. In the great majority of instances the disease becomes chronic if not placed under treatment. In some Eastern countries special hospitals have been provided to care for beriberi paralytics. If treatment and proper diet are provided, even after the paralysis has extended over a period of some months, recovery usually takes place, but it is very slow. There is apparently no immunity conferred by one attack of the disease. In addition to those actually ill with the disease, there are many others who have only slight symptoms, which are not sufficient to prevent them from pursuing, in a handicapped way, their usual avocations.

Beriberi patients are especially likely to contract malaria, dysentery and tuberculosis.

The sequelæ of beriberi depend very largely upon the stage of the disease at which effective treatment is administered. If the degeneration of the nerves has lasted over a period of years, many sufferers of beriberi become permanently paralyzed, especially in the lower extremities, and are unable to walk.

The *diagnosis* may be based upon the loss of knee-jerks, tenderness on pressure over the epigastric region, patches of hyperesthesia, and, later, anesthesia of the legs, pain on pressing the muscles of the calf, and of the arms, pretibial edema, absence of albuminuria and absence of fever. Numerous cases are met with among persons who have been living on a one-sided, unbalanced, monotonous ration. Confusion is prone to occur in differentiating beriberi from arsenic, lead and other poisons, and in such instances the differentiation is sometimes difficult, and generally depends upon the dietary history and the occupation of the patient. Argyll Robertson pupil helps to separate it from locomotor ataxia, and also the Wassermann and luetin tests. Uncinariasis is frequently confused with beriberi, but in this quandary the anemia and the presence of the parasites make a sharp distinction.

The mortality of the disease varies very greatly, and probably depends upon the stage of the disease during which the patient comes under treatment. The mortality has been given at from 2 to 60 per cent. In the pernicious forms the disease is nearly always fatal. The avoidance of cardiac strain is an important point in a favorable prognosis.

TREATMENT.

Treatment should consist of rest in bed and extreme precaution to prevent cardiac strains. In recent years the administration of an extract made from the polishings of rice has been used with apparent success. Guerrero[45] has reported a series of 447 cases treated with the rice extract with a mortality of 5.59, whereas the control series of 349 in which the rice extract was not used showed the mortality of 7.16. This extract, according to Vedder,[46] is made as follows:

"Rice polishings, or tiqui-tiqui, may be obtained from any rice-mill, but should perferably be from a recent milling. The finest grade of polishings should be carefully selected, since some of this product is very coarse and consists mostly of hulls. The tiqui-tiqui is first sifted to remove hulls and weevils. Gauze of about seven meshes to the centimeter is used for this purpose. This fine powder is weighed and mixed with 90 per cent. alcohol in the proportion of 3 liters (quarts) of alcohol to each kilo of polishings. It is then allowed to macerate for twenty-four hours. A glass jar or white enameled receptacle serves for this purpose, and the mixture should be repeatedly stirred or shaken, since the tiqui-tiqui sinks rapidly to the bottom, forming a densely packed mass which the alcohol penetrates with difficulty. During the extraction the alcohol becomes of a deep-green color, due to the fat that has been dissolved out. At the end of twenty-four hours the alcohol is siphoned off and filtered until absolutely clear. Since a very considerable quantity remains in the tiqui-tiqui, this should be squeezed in a press or washed with fresh alcohol, and the residuum filtered and added to the alcoholic filtrate already obtained. The extraction should then be repeated several times, again using 3 liters (quarts) of alcohol to each kilo of polishings. This is neces-

sary because neuritis-preventing substances are only slightly soluble in cold 90 per cent. alcohol, and experience has shown that if the polishings are not repeatedly extracted the full therapeutic action of the polishings is not obtained. The combined alcoholic filtrate is then placed in a water bath provided with a thermometer, and an electric fan is arranged so as to throw a strong current of air on the surface of the alcohol. As a result of the heat and the movement of air the alcohol repeatedly evaporates. It is essential that the temperature of the extract should not be permitted to rise above 80° C. (176° F.), since extended observation has shown that greater heat is liable to decompose the active neuritis-preventing principle. Whenever the temperature of the extract approaches 80° C. (176° F.) the fire should be extinguished until the temperature drops. This process is continued until all the alcohol is evaporated. The residue is poured into a separating funnel and allowed to stand for about an hour, when it will be observed that the liquid has separated into two layers. The upper and larger portion is of a deep-green color and consists of the fat. The lower and smaller layer is brown in color, of syrupy consistency, and contains a number of substances that have been extracted by the alcohol. This lower layer is carefully drawn off, leaving the fat behind. It varies in amount, but about 25 mils (6.7 f℥) usually will be obtained from each kilo of polishings. The brown syrupy fluid so obtained from 1 kilo of polishings is diluted to 60 mils (2 f℥) with distilled water, whereupon a heavy precipitate is formed. This precipitate consists of substances that were soluble in alcohol, but are insoluble in water. After allowing the mixture to stand for a while the precipitate settles and the clear fluid is filtered off. This filtrate constitutes the extract as we have used it. Each 60 mils (2 f℥) contains the substances that have been extracted by this method from 1 kilo of polishings."

Guerrero[47] recommends that 15 mils (½ f℥) of an extract composed of 1 part to 15 should be administered to a child in the course of twenty-four hours, and proportionate doses for an adult. He recommends in acute cases that 5 mils (1.35 f℥) of extract should be administered every half-hour until the acute symptoms subside. The diet is of the greatest

importance in the treatment of the disease. In the Eastern countries it has been found of great value to use a bean known as the *Phaseolus radix* (often called Mongo in the Philippines) in such quantity as the patient can readily digest. The use of ordinary yeast in teaspoonful doses at intervals of every three or four hours is reported to be of value. Beans, peas, meat and whole bread are also indicated. Tonics such as iron, quinin and strychnin should also be used. As soon as the acute heart symptoms subside in cases which suffer from paralysis of the limbs, a certain amount of exercise should be insisted upon. In some hospitals this is accomplished by stretching a rope at the height of about 5 feet along a veranda or ward, and having patients walk up and down and steady themselves with the rope. Electricity and massage are also valuable adjuvants. If rice-extract in acute cases is given promptly, it will not often be necessary to make use of strychnin; however, if the extract is not available, active stimulation should be employed in the same manner as in other cases of cardiac failure. Rice should be eliminated from the diet.

Various measures have been suggested for making effective the knowledge as to the manner in which beriberi may be prevented. A campaign of education has been most persistently carried out in the Philippine Islands during the past four years, principally through teaching some 400,000 pupils in the public schools. Unfortunately, even after the public was prepared to use the unpolished rice, it has been impossible to obtain it, because the rice-mill managers did not care to change their process of manufacture. Also, it so happens that the rice-exporting countries are not always the ones whose inhabitants subsist on rice and fish, and as their own population is often not affected, they do not take any great interest in preventing the disease in other countries. The Far Eastern Association of Tropical Medicine, at its meeting held in Manila in 1910, passed a resolution calling the attention of all governments concerned to the desirability of bringing about the use of unpolished rice. This resolution was again reaffirmed at the meeting held in Hong Kong in January, 1912. The International Congress of Medicine and Surgery held in London during August, 1913, substantially

reiterated the resolutions of the Far Eastern Association of Tropical Medicine referred to above. The knowledge of the prevention of beriberi has now been available for over five years, and so far there has been practically no application of this knowledge outside of government institutions. To sum up, education has been faithfully tried in one country, and has made very little progress. Inducing persons to add other constituents to their ration, which would give them the substance that is missing from polished rice, has been tried in Hong Kong, and has only succeeded in the case of persons in hospitals or jails. In Sumatra, notwithstanding the efforts of the medical profession, little progress has been made. In view of these failures the following is suggested:

Place a tax of, say, 2 cents United States currency (1 penny) per pound on polished rice. United action by the British Colonies would, in all probability, result in other countries interested adopting similar standards. Rice-mill owners would immediately find it desirable to make unpolished rice, and the use of polished rice as a staple article of diet would soon cease. There would be no additional burden placed upon the ordinary consumer, and the fiscal arrangements in all countries concerned with regard to rice would remain *in statu quo*. At the same time, it would not deprive those who use European diet from obtaining at a small increased cost the polished rice which they prefer, and which, owing to their varied diet, does them no harm.

The classification of rice into polished and unpolished rice can be based upon a simple chemical test. Phosphorus pentoxid (P_2O_5) is a safe indicator as to the degree of polishing and as to whether a rice contains a sufficient amount of the beriberi-preventing principle. It has been ascertained by experiments that rice which contains 0.4 per cent. or more of phosphorus pentoxid will not cause beriberi, and, therefore, may be called unpolished rice, whereas rice containing a lesser amount of phosphorus pentoxid may cause beriberi and be classed as polished rice.

It is quite possible that there may be serious objections on the part of the people directly affected by having their white, polished rice changed to a darker, unpolished variety. This difficulty could probably be overcome by education and

by inducing the millers to produce a clean, partly polished rice which would be acceptable to the people, and yet not cause beriberi.

Low-grade Beriberi. In this form of the disease the symptoms are but slight. The ration is probably only slightly deficient. Its presence may go unsuspected for a long time. The patient has a number of indefinite symptoms. Often there is slight pain in the abdomen, especially on pressure. There may be pain in the calves of the legs. Palpitation of the heart on slight exertion is common. At times the presence of beriberi is discovered in mothers of nursing infants when the health of the mother, and especially that of the child, improves upon administering the extract of rice polishings to the mother.

Infantile Beriberi. In the Philippines infantile beriberi is known also by the terms Taon and Taol Suba. For some time it has been suspected a large percentage of the abnormal infant mortality, especially in the Philippines, is closely associated with beriberi in the mother. It manifests itself probably in the form of infantile beriberi, or other condition that may be ascribed to faulty nutrition among breast-fed infants. This seems all the more probable when it is remembered that the mortality among breast-fed infants in the Philippines is about double that of bottle-fed children. In Europe and in America the reverse is true, viz.: the mortality is always lower among breast-fed infants. Special investigations of this question have been made by McLaughlin, Andrews, Vedder, Guerrero, Albert and others, but the number of cases studied and the amount of data presented have not been regarded as sufficient to draw definite conclusions. As over half of the infants born in Manila die before they reach 1 year of age, it will be apparent that this is a matter which affects many thousands of lives, and additional information is most desirable before recording definite conclusions.

A striking example of this form of the disease is seen in the mothers of nursing infants who use an unbalanced diet. Children who are cross, cry, fail to sleep and are emaciated often improve in a miraculous manner when an extract of the cortical layer of the rice is administered to the children or even to the mother who feeds them by the breast.

TRYPANOSOMIASIS.

Sleeping Dropsy, Trypanosome Fever, Negro Lethargy and Morbus Dormitious are common synonyms for the various types of acute and chronic infections due to trypanosomes described by Dutton, Stephens and Fanthan.

The *Trypanosoma gambiense* of Dutton[48] is chiefly spread by the *Glossina palpalis.* The *Trypanosoma rhodesiense* of Stephens and Fanthan[49] is spread by the *Glossina morsitans.* The trypanosomiases are characterized by an inflammatory condition of the lymphatic system leading to encephalitis.

Mention is made of "Sleeping Sickness" in the book entitled "Navy Surgeon," by John Atkins, published in 1734. He describes a sleeping "distemper" which he found among the natives of the Guinea Coast in 1721. Since that time the disease has been frequently mentioned in the literature. It was not, however, until 1901 that Ford and Dutton[50] found a trypanosome in the blood of a patient suffering from a peculiar type of fever. In 1901 Castellani,[51] in Uganda, found a trypanosome in the spinal fluid of a person suffering from sleeping sickness. Later Law and Castellani[52] called attention to the two constant symptoms, namely, fever and the peculiar tremors. In 1903 Sir David Bruce and Navarro[53] showed that the disease was spread by a *Glossina palpalis.* Kleine,[54] in a series of important researches, showed that the micro-organism undergoes a cycle of development in the fly, which was confirmed by Sir David and Lady Bruce.[55] In 1907 a bureau under the British Colonial Office for the study of the disease was founded. In 1910 Stephens and Fanthan[56] created a new species.

The disease is almost chiefly confined to the West Coast of Africa and Sierra Leone. Trypanosomes have been frequently carried to other tropical regions, but probably owing to the absence of the special fly necessary to its development the disease has not spread. Cases are frequently introduced into England and the United States, but no dissemination has been noted as a result of this experience. Trypanosomiases are probably due to several species of trypanosomes, but the *Trypanosoma gambiense* and the *Trypanosoma rhodesiense* cause the more common types of the disease. In South

America, specially in the State of Minas in Brazil, a trypanosome fever occurs which is due to the *Trypanosoma cruzi Chagas,* which is spread by the bug *Lamus megistus* (Burmeister). The symptoms of the disease are entirely distinct from African trypanosomiasis. One type resembles myxedema, another pseudomyxedema. Many cases have hypertrophy of the lateral lobes of the thyroid gland. It is frequently confounded with severe cases of uncinariasis. The disease is very rare, and scarcely warrants a full description in a book of this kind.

The *pathology* of the several trypanosomiases appears to be restricted to a chronic inflammation of the lymphatic system, which may be due to the toxin elaborated by the organism. The trypanosome either enters the lymph-stream of the human host after the bite of the fly, or it is blocked in the lymphatic gland into which it passes. It is thought that perhaps the organism may find its way into the blood-stream, and then by rupture of a capillary it may reach the lymph-channels.

Prevention measures must be based upon the destruction of the *Glossina palpalis* and the *Glossina morsitans.* To prevent these flies from having access to sources of infection like human beings, or to animals that harbor the disease, is the next step in order of importance. Wild animals probably are reservoirs of infection. Duke[57] has found the *Trypanosoma gambiense* in two marsh antelopes. Up to the present time infected flies have been found only in Africa within a few degrees of the Equator. The greatest precautions should be taken with persons infected with the disease who come into regions in which the tsetse fly occurs, to the end that new centers of infection may not develop.

The course of the disease readily divides itself into three stages: the incubation, the febrile or glandular, and the cerebral. The *incubation period* is not known. Various authors agree that the disease is most likely to occur within from ten days to two weeks after the bite of the fly. In Europeans the onset of the disease begins with fever associated with an erythematous eruption. The fever, which may be intermittent or remittent, lasts for about a week, and then disappears without treatment, only to occur for the same or much longer

periods. The eruption in Europeans begins with badly defined pinkish patches, which gradually fade in the center until only a margin, or ring, is left. The eruption may appear upon any part of the body, although it is most frequently found on the trunk. It is very difficult to recognize the typical erythema in negroes.

After a *febrile stage,* which may have lasted for some weeks, months, or sometimes even years, a change becomes noticeable in the habits and disposition of the patient. Those who previously have been active show disinclination for exertion and prefer to sit quietly or lie down. This is followed by careless habits and difficulty in walking. Then the actual sleeping stage begins. The sleep may be excessive, but it is not necessarily the prominent symptom so often described. It is more of a lethargy from which the patient can be roused, and, despite the obvious somnolence of the subject, questions are answered intelligently. Fine tremors may be noticed first in the tongue, and later in the hands, arms, and sometimes in the legs; occasionally the tremors are so severe that the whole body shakes. There is usually fever, the temperature rising in the evening from 100° to 104° F. (37.7° to 40° C.). The gait is peculiar, there being apparent difficulty in raising the feet, but there is seldom paralysis; superficial reflexes are normal at first; the deep reflexes may be increased, and then completely lost; there is frequently a gradual reduction in the number of erythrocytes to 2,000,000 per cubic millimeter, or even less, although occasionally a case is encountered in which there is an increase in the number of red cells. The demonstration of the trypanosomes in the blood furnishes conclusive evidence of the presence of the disease.

When the disease is due to the *Trypanosoma rhodesiense* it usually runs a rapid course, and is generally fatal within four or five months. When due to the *Trypanosoma gambiense* it may last a number of years. Complications such as malaria, filaria, intestinal parasites, and cerebrospinal meningitis are common.

The *diagnosis* depends upon finding the trypanosomes in the blood. The endemic areas in which the disease is found; the fever which does not yield to quinin; the fine tremor of

the tongue, and the drowsiness, the apathy, and later the generalized tremors are significant.

The prognosis in untreated cases is nearly always fatal. Recently considerable success has been had in the treatment of the disease with various preparations of arsenic, and especially atoxyl.

TREATMENT.

Manson's method of administering atoxyl in 2- or 3- grain (0.13 or 0.19 Gm.) doses by intramuscular injection every third day for two years offers the best hope of recovery. Some patients show considerable chemoresistance, which is apparently due to the fact that trypanosomes become resistant after a prolonged use of the same drug. On this account various modifications of administering the arsenic have been suggested. It has been found useful to supplement the atoxyl with 2 grains (0.13 Gm.) of sodiotartrate of antimony dissolved in 2 pints (1 l.) of water and administered by mouth or rectum. Mercury has also been used with apparent success. If there is coincident malaria or infection with intestinal parasites, appropriate treatment also must be given for these.

HOOKWORM INFECTION.

Hookworm infection is an infestation of the human alimentary tract with small nematode worms which, in many instances, are responsible for severe anemia and for other constitutional disturbances. Under the term hookworm have been included the infections caused by *Ancylostoma duodenale* (Dubini) and those due to the *Necator americanus* (Stiles). In the Eastern countries it is customary to refer to infections due to either of these worms as ancylostomiasis, although a strict definition of the terms would restrict ancylostomiasis to infections caused by the ancylostome. On the Western Continent it is customary to refer to either of the infections as hookworm disease or uncinariasis.

There are indefinite references to a disease which may have been due to hookworm infection as early as 1550 B. C. In the New World, Piso, in 1648, in his history described a fatal disease in Brazil which may have been uncinariasis.

Frequent references occur to a disease resembling uncinariasis by various authors of books relating to Santo Domingo, Jamaica, British Guiana, and as early as 1808 in the United States. It was, however, not until 1843, when Dubini, in Italy, found the *Ancylostoma duodenale* as the cause of miners' anemia, that the diagnosis of the disease was placed upon a definite basis.

Bilharz[58] recognized the disease in Egypt in 1852, Wucherer[59] in 1872 in Brazil. In 1902 Stiles[60] described the *Necator americanus* found in the Southern States as a new species of the worm. In the same year Boycott and Haldane[61] found the disease among miners in Cornwall, England. In 1898 Loos traced the method of infection through the skin, blood-vessels, lungs, trachea and esophagus to the intestinal tract. In 1899 Ashford[62] drew attention to the great prevalence of the disease in Porto Rico, and the high eosinophilia. Soon afterward a large number of examinations made in Porto Rico by Ashford,[63] and in the United States by Stiles,[64] showed that there was a high incidence of infection among the residents of Porto Rico and of the Southern States of the United States. These writings attracted much popular attention, and public-health measures were undertaken on a large scale for the control of uncinariasis. These measures were particularly difficult of application in the United States, because of the attitude that it was rather a reflection on a community to admit the presence of this disease. This feeling was accentuated by the fact that the disease was associated with clay-eating, and also because the Press frequently referred to the infection as the lazy worm. The excellent work done in Porto Rico, however, soon resulted in convincing the public of the great importance of the disease, and of the improved health which followed when measures for its control were actively undertaken. It was soon very generally recognized that hookworm infection was intimately associated with anemia, and especially the very debilitating disease known as tropical anemia.

In 1909 the Rockefeller Sanitary Commission was organized with a fund of $1,000,000, for the purpose of attempting the control and the relief of the disease in the Southern States of America. Practical work was begun in 1910 in

co-operation with State and local health departments or other officials where no health officers were available, and the success achieved, especially in winning popular approval, not only for measures directed against hookworm infections, but also for public health measures in general, has resulted in one of the notable advances in the public-health movement which is now making such rapid progress throughout the civilized world. In 1913 the International Health Commission was organized under the Rockefeller Foundation. It has for its object the relief and control of hookworm infection throughout the tropical world. The enormous magnitude of this plan may be well appreciated when it is recalled that hookworm disease occurs in practically all countries at altitudes below 3000 feet and between 36 degrees north latitude and 30 degrees south latitude.

Hookworm surveys made during the past few years throughout the world show that the disease is even more prevalent than was at first anticipated. Recently (1916) some thousands of examinations made in Ceylon showed an infection of 96 per cent. It is very rare not to find at least a 10 per cent. infection. The percentage of the infection may be said to depend upon the effectiveness with which human excrement is disposed of. A great feature in the introduction and spread of hookworm disease into areas heretofore free from infection has been the emigration of labor from India. For instance, the migration of the Tamils from Southern India to the West Indies, Ceylon, Federated Malay States, and the Fiji Islands has resulted in an infection rate which is probably higher than existed in the sections of India from which they came. This is largely traceable to the fact that these emigrants are generally employed in agricultural operations, and are closely housed in barrack buildings, which are seldom supplied with adequate latrine accommodation. This has resulted in much concentrated soil pollution, and as these laborers practically always go barefooted, there is practically no obstacle to their contracting repeated hookworm infections. The disease is confined almost entirely to a belt around the earth inclosed between latitude 36° north and latitude 30° south, but even in this tropical belt there is a marked reduction in the incidence of infections when an altitude of

3000 feet and above is reached. In many high altitudes the disease is not found, and this comparative freedom of infection is probably due to the fact that the lower temperatures (cooler weather conditions) which prevail at higher altitudes are inimical to the development of the larval stage of the hookworm. However, hookworm infections often do occur at high altitudes, and the small amount of infection found may be due to the lack of introducing the infection. It is worthy of note that there are large tropical districts in which the disease does not exist, or at least prevails to a very slight extent, examples of such districts being the Bombay Presidency and the Punjab in India. At first sight this might appear to be due to the hot, dry conditions which prevail, but under similar conditions in Egypt, where it is equally dry and hot, 50 per cent. hookworm infections have been found. It is reasonable to assume that much of this difference in the infection rate may be due to the safer method of human excrement disposal, and to the small number of infected persons who are introduced into the Punjab.

The disease is caused by the presence of the *Ancylostoma duodenale* or the *Necator americanus* in the intestines. So far as known, these parasites live only in human beings. The *Ancylostoma ceylonense* is commonly found in dogs, but there is no evidence to show that they may live in the human intestines. The cause of the symptoms which the disease produces has not yet been satisfactorily demonstrated. Some authors believe that the symptoms are due to toxins set free by the embryo in its travels from the skin to the alimentary canal. Others believe them due to toxins given off by the adult worm after it has reached the intestines.

The points at which the embryos entered the skin in sufficient numbers usually show an eruption of papules or vesicles, which, in all probability, are due to skin infections by bacteria introduced through the larvæ. The skin lesions are called Ground Itch in Assam and other English-speaking countries, and Mazamorra in Spanish countries. It has been shown that the entrance of the larvæ is attended by itching and a macular eruption, followed the next day by swelling of the part. Usually five days afterward there is an enlargement of the lymph-glands of the affected areas, and the eruption usually

disappears by the twelfth day. Observers in many countries have reported that they are unable to associate ground itch with hookworm infection. It may be stated, however, that when careful observation is possible, ground itch is usually found. The patient usually fails to recall the eruption, which fact is probably due to the long time which elapses between the eruption and the appearance of symptoms. The ova usually appeared in stools six weeks after the original infections took place.

Still others believe that the symptoms are due to the anemia caused by the hemorrhages produced by the parasite when it feeds on the walls of the intestines. It was also thought that perhaps the infected wounds resulting from the bites of the parasite may be responsible for a toxemia which causes destruction of the erythrocytes.

There is not much evidence of racial immunity, except in so far as it concerns the custom of going barefooted or other habits that lead to infection. In Porto Rico, for instance, there was an infection of 71 per cent. among the Europeans, 54 per cent. among mulattoes, and 41 per cent. among negroes. In Ceylon the reverse held true, there being a 90 per cent. infection among Tamils and 20 per cent. infection among Europeans. It seems quite probable that these differences can be reconciled by the difference in the soil pollution and personal habits of the various races, in the particular place in which the observation was made.

There is no general agreement as to whether the larvæ produce symptoms in the patient during the passage from the skin to the alimentary canal, but soon after the worms reach the alimentary tract in sufficient numbers marked reductions in the hemoglobin and other blood changes take place. The anemia is ascribed by Castellani[65] to "hemolytic toxins secreted by the worm, actual loss of blood from the bites of the worm, and microbic secondary infections."

Edema of the feet and a white appearance of the conjunctiva may be noted on inspection of the body. The intestines are pale, the peritoneum is saturated and heavy, and some straw-colored fluid is generally found in the peritoneal cavity. All of the organs appear damp and pale. The lungs are edematous, the heart is pale and fatty, and sometimes there

is hypertrophy of the left ventricle. The liver is fatty. The spleen is usually shrunken. The pancreas and super-renal glands are normal. The kidneys are often enlarged, pale and fatty. The jejunum and ileum frequently show ecchymotic areas which were caused by the bites of the hookworm. The stomach may show evidence of chronic indigestion. Hookworms varying from a few to as many as 3000 are found in the jejunum and ileum.

The *Ancylostoma duodenale* has a cylindrical body which tapers from the back to the front in both sexes. During life it is pinkish red in color. The mouth is terminal. On its under side it has two pairs of hook-like teeth, and on the upper or dorsal side one pair of teeth. The male measures about 10 millimeters (⅖ in.) in length and 0.4 to 0.5 millimeter (0.016 to 0.02 in.) in breadth. The female measures 12 to 13 millimeters (0.48 to 0.52 in.) in length, and has a vulva at the junction of the middle with the after-third of the body. The male generative apparatus consists of a testis in the form of a tube, an oval vesicula seminalis, and a gland which exudes a sticky substance to fix the male to the female during conjugation.

The *Necator americanus* has a cylindrical body which is somewhat smaller in diameter at its anterior part. The head is usually bent acutely on its dorsum. The mouth has a ventricular pair of prominent, semilunar, chitinous plates and a dorsal pair of plates slightly developed. In the floor of the mouth the dorsal head gland-opening resembles a conical tooth, and deeper in the cavity there are a pair of dorsal and a pair of submedian lancets. There is an excretory pore at 0.5 millimeter (0.02 in.) behind the mouth. The male measures from 7 to 9 millimeters (0.28 to 0.36 in.) in length and from 0.3 to 0.5 millimeter (0.012 to 0.02 in.) in breadth. The female is 9 to 12.6 millimeters (0.36 to 0.504 in.) in length.

Practically, the two parasites may be readily distinguished, first, by their size, and, second, when they are dropped into hot alcohol, the *Ancylostoma duodenale* resembles an elongated letter S, whereas the *Necator americanus* resembles a cane with a crook upon it, as shown in the accompanying illustration.

The adult worms are chiefly found in the jejunum of the

human host, where they feed upon the villi. Here the females lay their eggs, which are oval in form, with broad, rounded poles surrounded by a colorless shell which incloses a granular mass separated from the shell by a considerable space. As the eggs travel down the intestinal tract the granular mass divides into two and usually into four segments, which is the condition in which the egg is usually found in the feces. The rapidity of the development in the feces depends upon the temperature of the atmospheric air. When conditions are favorable as to air, water and heat, the embryo may be seen coiled up in the egg twenty-four hours after it has been voided. It then escapes as a larva and feeds on fecal matter. The larva is needle-shaped, running to a point posteriorly, and measures from 200 to 250 microns in length. In the



Ancylostoma duodenale.



Necator americanus.

tropics at the end of five days it ceases to grow, and, feeding on feces, it sticks to the water or moist earth, where it may remain unchanged for months, living on the food-matter inclosed in its own cell. This represents the encysted stage. The larva may become quite active and swim and climb on wet surfaces. During this period it is ready to infect man, which it does usually through the hair-follicles in the skin, causing eruption or sores commonly called ground itch. From the hair-follicles, according to Loos,[66] it forces its way through the subcutaneous tissue into the veins and lymphatics. Those which have entered a vein go to the right heart and thence to the lungs. If the larvæ have entered the lymph-channels many are killed in the lymphatic glands, but some get through to the blood and are carried to the lungs. In the lungs they penetrate the capillaries, reach the air-cells, travel up the bronchi to the trachea and larynx, and then descend the esophagus. The time occupied by this migration is generally believed to be ten days. After the fourth day ecdysis takes place in the alimentary canal, which change occurs from eight

to ten days after their arrival; they then measure 3 to 5 millimeters (0.12 to 0.2 in.) in length. Eight days later the generative organs begin to attain maturity, and then first copulation takes place. A few days later eggs appear in the feces, so that from the time infection in the skin took place until eggs are found in the human stool a period of four to six weeks elapses.

Sambon[67] believes that some of the larvæ may follow the route indicated above, but that infection also takes place by the larvæ passing from the pulmonary artery to the pulmonary vein, whence they are carried by the blood to the jejunum, which they penetrate and enter the lumen of the bowel. Sambon also believes that when larvæ are taken directly into the mouth they probably pierce the walls of the esophagus and reach the intestinal tract by the way of the blood-vessels, in exactly the same way as if they had originally penetrated the skin.

Usually the first *symptom* is a dermatitis of the feet and legs, and when it affects the sole of the feet it is commonly known as sore feet of coolies and also as ground itch. Considerable itching is generally present in these lesions. The onset of the disease in the great majority of instances is most insidious. Many persons are afflicted with hookworm infection without having enough symptoms to attract attention. Slight digestive disturbances are common. Others become anemic a few weeks after the parasites reach the intestines, and leucocytosis with eosinophilia is the rule. Anemia is probably the most common symptom. The pearly whiteness of the conjunctiva and the white finger-nails are characteristic, and when once seen are seldom forgotten. The peculiar suffusion of the countenance soon leads even laymen to suspect the disease. The feet and ankles are dropsical. There is usually emaciation, but this is often concealed by the dropsy. In many cases there is only edema of the feet, while in others it may extend to the legs, scrotum and face, and be associated with ascites. In the Fiji Islands a black tongue has been associated with hookworm infection, but examinations of natives who were not afflicted with hookworm disease showed black tongue in almost similar proportions. Often there is chronic dyspepsia, due to gastritis, and marked

by nausea and pain over the stomach. There may be diarrhea or constipation. There is usually a marked reduction in the percentage of hemoglobin, which sometimes drops to 10 per cent. before the patient succumbs to the disease. The blood condition often resembles that found in chlorosis. The blood-picture is almost the reverse of that found in pernicious anemia. The eosinophilia averages about 10 per cent. In a well-marked case the erythrocytes may be reduced to a million, with a normal leucocyte count. Patients frequently complain of palpitation of the heart and difficulty in breathing. The vessels of the neck are frequently seen to palpitate in a very marked manner. The pulse is usually quick, weak, thready, dicrotic, and sometimes intermittent. In children enlargement of the liver is frequent. In many cases there is a low intermittent type of fever in which the temperature seldom rises to about 38° C. (100.5° F.). Some cases present various modifications in the temperature feature, and frequently it is difficult to distinguish hookworm fever from malarial fever, kala-azar and trypanosomiasis. The urine is usually copious, pale, and often alkaline, with a specific gravity varying from 1.010 to 1.015; albumin is rare, but there may be an increase of indican and urobilin.

Mental and physical hebitude may be marked, and this has frequently been referred to in the popular press as a symptom due to the lazy worm. It often happens that laborers who have rested are quite capable of performing their ordinary tasks on Monday, being less able on Tuesday, and by Wednesday afternoon they are completely exhausted, and must rest until the following Monday before being able to resume their tasks. When anemia becomes extreme, death may result from heart-failure; but it not infrequently happens that death is due to some intercurrent affection.

A definite *diagnosis* of hookworm infection depends upon finding the ova or the adult worms in the stools. The following method has recently been devised by Barber,[68] and apparently resulted in finding the hookworm eggs with greater certainty than is the case by the ordinary microscopic technic heretofore employed.

Field workers investigating uncinariasis may desire to learn of a convenient method by which, without the aid of a

centrifuge, from 70 per cent. to 90 per cent. of the positive cases of hookworm infection can be expeditiously detected. Dr. M. A. Barber, who has furnished the following particulars of the method, recommends its use, when it is anticipated that the percentage of positive cases will be high (80 per cent. to 90 per cent.). He estimates that by its use, together with that of the centrifuge in the remaining apparently negative cases, 100 to 150 specimens can be examined per diem by one microscopist.

The details of the method, which depends primarily on the different specific gravities of the hookworm ova and a mixture of glycerin and saturated NaCl, are as follows:

1. The feces (preferably a fair quantity) in a convenient container, such as a half cocoanut-shell or latex cup, are thoroughly stirred with a small flexible stick, to obtain an even distribution of ova through the mass, water being added if necessary, to form a smooth, soft paste.

2. Slides either 3 x 1 or 3 x 2 inches (7.6 x 2.5 cm. or 7.6 x 5.0 cm.), properly labeled, are prepared by drawing around the margin of the upper surface a broad line with a grease pencil or paraffin.

3. A small portion of the fecal mass is placed upon the slide, and, with the aid of a small stick or similar appliance, is thoroughly stirred into a mixture of equal parts of glycerin and saturated NaCl solution dropped on to the slide. The mixture on the slide should be of such volume as almost to overflow the greased edge. The surface of the fluid will be convex; the aim is to add as much fecal matter as possible without making the specimen too opaque for microscopic examination. Practice soon shows the degree of opacity which should not be exceeded.

4. The slides now ready for examination are placed upon a slideholder, which holds from ten to fifteen slides, a rough sketch of which is given herewith.

The preparations are examined without a cover-glass. Owing to the higher specific gravity of the glycerin and salt solution, the ova float to the surface, and are easily detected with a low-power lens.

The objective should be focused to the surface level and not to the depth of the fluid. The preparation should be

examined within an hour or so, otherwise hyaline ova may become too transparent for easy recognition.

5. In the event of the first centrifuge search of the apparently negative specimens proving negative in any case, a second fecal specimen obtained on a subsequent day should be examined.

Observers who have had considerable experience with hookworm disease soon acquire the ability to make reasonably accurate diagnosis by the typical facial appearance. This is difficult to describe, and probably can only be learned by practice. Some of the principal points concerned, however, are the muddy complexion in whites, the suffused features, the extreme whiteness of the conjunctiva, and the tired expression. The foregoing facies associated with indefinite



Fig. 11.—A thin board large enough to carry ten slides or more is used, small cleats ½ in. x ½ in. (12.7 x 12.7 mm.) wood being fixed at both ends of the underside of the board. This enables the holders to be stacked if desired.

abdominal pains, swelling of the feet, extreme pallor of the finger-nails, and slight irregular fever should always be followed by a search for adult worms, and by a careful microscopic examination of the feces for hookworm eggs. The adult worms can be recovered readily by washing the stools through cheesecloth or fine muslin.

TREATMENT.

The number of treatments which have been advocated are legion. During the past few years scientific field work has done much to bring order out of the chaos. The Hookworm Board of the Rockefeller Foundation (Darling, Barber and Hacker), which has been working in the Federated Malay States during 1915 and 1916, has made careful tests of many remedies which have been advocated. It has come to the conclusion that oil of chenopodium shows an efficiency of 92 per cent. as compared with 82 per cent. for thymol, 60 per

cent. for betanaphthol and 40 per cent. for eucalyptus. The chenopodium method used closely resembles that advocated by Shüffner, Kunen, Vervoort and others in Sumatra, who have reported the treatment of over 300,000 cases. The method used by the board of the Malay States consists of giving the patient liquid diet and a cathartic dose of magnesium sulphate, next morning no breakfast, but a cup of tea may be permitted. At 7 o'clock a gram (15.4 grs.) of oil of chenopodium, followed by a similar dose at 8 o'clock and at 9 o'clock, or a total of 3 grams (46.3 grs.), and at 11 o'clock a cathartic dose of magnesium is given. It has been found that the dead worms continued to be voided for several days, and usually 80 per cent. of the total is voided by the second day, and up to 93 per cent. on the third day. Many instances have been reported in the literature of oil of chenopodium producing toxic effects, but in such instances usually a much larger dose than the foregoing has been administered. There is also much reason to believe that chenopodium may have a cumulative effect, and it is considered very unwise to administer the treatment continuously for a number of days, even in small doses. Purgative action after chenopodium has been given is essential. After a treatment has been given it should not be repeated for at least ten days. The dose given above should be reduced proportionately for children. There are, however, still many advocates of the thymol treatment. The system used by the various commissions working in many countries in co-operation with the International Health Board of the Rockefeller Foundation is as follows:

"1. On the day preceding the treatment give a large dose of sulphate of magnesia at 5 P.M. No supper should be eaten. The saline should thoroughly empty the alimentary canal.

"2. Remain in bed the following morning without food.

"3. At 6 A.M. take one-half of the thymol; at 8 A.M. take the remainder.

"4. Take a large dose of sulphate of magnesia at 11 A.M. This should be repeated if a thorough movement of the bowels is not secured within two hours.

"5. No food should be taken until after the bowels have moved thoroughly, and then no greasy foods, milk, alcoholics or malt drinks should be indulged in.

"6. The usual diet and habits may be resumed on the day following treatment.

"7. If the patient feels weak or dizzy during the treatment give ½ cup of strong coffee without sugar or milk.

"8. Careful examination of the dejecta from the second dose of sulphate of magnesia should show the dead worms expelled.

"9. Several of these courses of thymol, given at weekly intervals, are usually necessary for cure."

Various methods have been suggested for administering the oil of chenopodium. Schüffner[69] has recommended dropping the oil into lumps of sugar; others have used gelatin capsules. Where large numbers of persons are to be treated, the capsule method is probably the most convenient, and there is no reason to believe that it is less effective than other methods of administration.

Prevention. In many respects uncinariasis is one of the diseases for which prophylactic measures may be applied with the greatest hope of success. The life history of the parasite is known; there is no multiplication in the human host; the propagation of the disease takes place only when hookworm eggs in human feces are deposited upon ground surfaces or in other places where later the resulting larvæ may come in contact with the human skin, or, perhaps, reach the mouth through food, water or other means. Briefly, then, multiplication of the parasite takes place solely outside of the human body, and there can be but the same number of adult parasites in the human intestines as there have larvæ penetrated the skin or entered the throat through the mouth or nose. The treatment of the disease is also very satisfactory, so that considerable progress toward preventing its spread can be made by freeing infected persons of the disease, and thus making their stools safe, even though excrement disposal is not satisfactory. Surveys made of the presence and spread of the disease in all parts of the world show that it exists in those tropical regions, and in mines and other places in temperate zones where there is sufficient warmth and moisture for the propagation of the larvæ which result from hookworm eggs in infected stools which have been allowed to cause soil pollution. Hookworm infection prevails, for instance, much

more extensively in small towns and rural districts than in large cities, this being due to the safer disposal of human excrement in cities. Again, the disease is found to prevail very extensively upon plantations in which there are large numbers of laborers who are closely housed and pollute the soil. There are many instances which show that through the introduction into plantation barracks of even a few persons who have hookworm disease almost the entire number of laborers soon become infected. On the other hand, with similar conditions, but with safe disposal of human excrement, there is no spread of the disease. Infection in mines and tunnels has been very general, especially in Europe, owing to the fact that no adequate methods for the disposal of human excrement were enforced. Because of the warmth and the moist condition of the soil and the fact that usually there is flowing water, the bottom surfaces of the mines or tunnels soon become thoroughly infected with hookworm larvæ, and even the wearing of boots, shoes, or other protection for the feet does not always prevent the larvæ from reaching the skin of the lower extremities. It is also possible that the drinking-water may have become infected. Some mines have been found remarkably free from hookworm infection, and in these instances examination of the water showed it to be heavily charged with iron sulphate, pyrites, or other chemicals. It has been found that ordinary cinders are inimical to the development of the larvæ, and for this reason cinders are often used in and around latrines, so that even if eggs are accidentally deposited their development will be extremely unlikely. Briefly, then, the prevention of the spread of uncinariasis may be accomplished by water-carriage latrines, by collecting excrement in deep pits, or by having it deposited directly into fly-proof pails, and then incinerated or buried in deep trenches. References have recently appeared in the literature in regard to the use of salt for the destruction of the hookworm eggs and larvæ in human excrement. Breinl[70] reports that this method has proven ineffective in actual practice. Iron sulphate in 1 per cent. solution has been reported upon very favorably. In brief, however, it may be stated that the method which gives the greatest hope of success is the proper collection and safe disposal of human excre-

ment, rather than dependence upon chemicals, or the disinfection of the stools or of polluted ground surfaces. Probably the best method of disposal of the contents of pails is by incineration. This may be most economically accomplished on plantations, for instance, by building cylindrical brick ovens of the desired capacity. A grate is placed inside the cylinder about 18 inches (45.7 cm.) from the ground; a suitable door is provided above it for the wood used as fuel, and another door below the grate for removing the ashes. The top of the cylinder is left open, and the contents of the pails are dumped into the top after the fire has been well started. Ordinary garbage and refuse may also be added, and assists in the burning. See illustration.

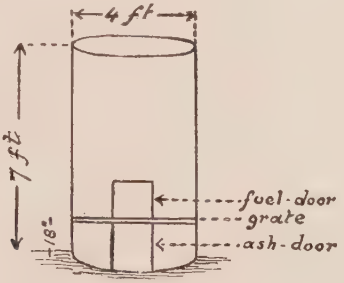


Fig. 12.—Incinerating oven.

Ovens of this kind have been used successfully in Borneo and other Eastern countries. An oven for the needs of one hundred people can be constructed for about $30.

THE DYSENTERIES.

Until very recent times it has not been possible to make any scientific distinctions between the different dysenteries. This was particularly true of amebic and bacillary dysentery, but the great progress in tropical medicine now makes it possible to classify them into those caused by animal parasites and those caused by bacteria.

The more common forms of dysentery due to animal parasites are:

1. Amebic.
2. Laveranic.
3. Leishmanic.
4. Ciliar.
5. Helminthic.
6. Balantidic.

Those due to bacteria are:

1. Bacillus dysenteriæ.
2. Hissbacillus dysenteriæ.
3. Shiga-Kruse.
4. Flexner.
5. Castellani.

Dysentery and Diarrhea. The term dysentery as distinguished from diarrhea signifies an inflammation of the bowel. Many authors distinguish between dysentery and diarrhea by the presence of blood in the stool of the former and by its absence in the latter. It is frequently referred to in English as bloody flux, in French as tenesme, in Italian as Flusso sanguigno, and in German as rhur. The name in native languages usually includes words which mean blood and mucus. Dysentery is a disease which has received attention by medical writers from the very earliest times. Even Hippocrates distinguished between motions which contained blood and those which consisted of other fluid matter.

Protozoal Dysenteries. Protozoal dysenteries are various forms of ill-defined dysenteries which are caused by protozoal parasites. Among these are the *Laverania malariæ*, the *Leishmania donovani, Balantidium coli, Balantidium minutum, Nyctotherus faba,* and a number of others. Of these the most important is that caused by the *Balantidium coli.*

BALANTIDIC DYSENTERY.

Balantidic dysentery is an acute inflammatory affection of the large intestine which may result in acute or chronic ulcers, caused by the *Balantidium coli* (Malmsten).

It is quite likely that these parasites were originally discovered by Leeuwenhoek, but Malmsten gave the first satisfactory description of them. Since then balantidial dysentery has been frequently recorded in medical literature, and the disease probably exists to a much greater degree in many countries than the literature would seem to indicate.

Cases occur frequently in the Philippine Islands, Japan and Europe, and isolated cases have been found in nearly all tropical countries in the Eastern Hemisphere in which a search has been made for the parasite by competent laboratory workers.

The disease is due to the *Balantidium coli,* but the method of transmission has not been satisfactorily demonstrated. A series of experiments made some years ago by workers in the Bureau of Science at Manila showed that *cholera, typhoid, Ameba histolytica, Balantidium coli,* and other micro-organisms fed to healthy pigs were not recoverable from the stools, with the single exception of the *Balantidium coli.* It is assumed that one of the principal factors in the transmission of this disease is due to soil pollution. Pigs probably become infected by consuming the stools of people that are afflicted with the disease.

The exact method of infection is unknown, but it probably differs very little from that of amebic dysentery.

The intestines often contain a diphtheroid membrane. Ulcers are generally found in the rectum and colon. The *Balantidium coli* can usually be found in the bowel contents and by scraping the mucosa of the infiltrated areas.

The disease usually attracts attention through attacks of severe diarrhea which alternate with constipation; there is also considerable disturbance of the digestion, and usually vomiting with loss of appetite occurs. Mucus is often found in the stools, but it is rare to find blood. Sometimes there is edema of the face and legs, and death may take place from exhaustion. The fever is usually higher than in amebic dysentery, especially during the period of acute diarrhea.

Sometimes the parasites cause cysts in the liver. The disease is usually associated with amebæ, uncinaria and other common intestinal parasites.

TREATMENT.

There is no specific treatment for the ordinary type of dysentery, and reliance must be placed upon rest in bed, liquid diet and active purgation with sodium sulphate. Rectal irrigations with quinin are often useful.

The same rules applicable to the prevention of amebic dysentery are also effective in guarding against balantidic dysentery.

AMEBIC DYSENTERY.

The use of the term amebic dysentery is now commonly restricted to an acute or chronic disease of the intestines, caused by the *Ameba histolytica* (Schaudinn), and exciting an enteritis terminating in characteristic ulcer formation. A frequent sequela is abscess of the liver and sometimes other parts of the body. It is known variously as amebiasis, amebic enteritis, amebic colitis and loschiasis.

As early as 1860 Lamb[71] observed amebæ in the stools of a child suffering from diarrhea. In 1870 Luis[72] found similar organisms in cholera patients, and in 1875 Losch[73] described amebæ which he recovered from the stools of a man suffering with chronic diarrhea. His drawings indicate that he was dealing with the tetragena. His observations were considerably obscured by the fact that later Grassi,[74] Cunningham[75] and Luis[76] found that the stools of healthy persons in the tropics frequently contained amebæ. Koch,[77] in 1893, found amebæ in ulcers of cases of dysentery in Egypt. Later Kartulis[78] began a series of investigations, and finally concluded that endemic dysentery was due to amebæ and epidemic dysentery was due to bacteria. Soon afterward a discussion began, which has not yet been finally settled, as to whether amebæ are always pathogenic or whether they are only pathogenic under certain circumstances, and as to the question of different kinds of amebæ. Schaudinn[79] insisted that the *Ameba histolytica* was the true cause of amebic dysentery, and other workers gave partial confirmation of Schaudinn's work. In 1913 Walker,[80] who made an extensive study of the disease in the Philippines over a period of several years, finally published a complete report in the *Journal of the Bureau of Science*. In this report practically all of the previous work was carefully reviewed, and most convincing evidence was adduced that there are pathogenic and non-pathogenic amebæ, but the histolytica was probably the ameba responsible for practically all cases of dysentery, and that the so-called ordinary water ameba could frequently be found in the stools of persons over period of years without the appearance of any symptoms; also, it was regarded as not likely that ordinary water ameba or other harmless types, by symbiosis or other

manner, could be transformed into a histolytica or other pathogenic ameba. He also showed that the tetragena is, in all probability, the same organism as the histolytica. Other workers throughout the world have practically come to the same conclusion. For instance, in Panama, in the installation of water supplies, the presence of the ordinary water ameba was disregarded, without any apparent deleterious results following therefrom. Walker also showed that pathogenic amebæ were, in all probability, not conveyed by water supplies in the ordinary sense.

Amebic dysentery is found very generally throughout the tropical world, but there are many large islands and other extensive areas with large populations in this belt in which the disease does not occur. It is frequently found in the Southern part of the United States, and recently cases have been found in New York City, the origin of which it is difficult to trace to outside sources. In the Tonga Islands, for instance, amebic dysentery is confined entirely to new arrivals who bring the disease with them. It is not known to exist in New Zealand, and no cases have been reported from the northern territory of Australia. It prevails extensively in Java, Malay Peninsula, Siam, Borneo, Sumatra, Philippines, China, Japan, Ceylon, and in many parts of India, Egypt and tropical Africa. It is said to be rare in Central America and the West Indies, but common in Brazil and Chili. In Europe it is known to occur in Russia and Germany, and, quite frequently, in Italy. The disease is often endemic, but never spreads in epidemic form.

In view of the recent work by Walker, in confirmation of that of Schaudinn and others, it may be said that amebic dysentery is caused by the *Ameba histolytica.* Although there are a great many kinds of amebæ, only a few are parasitic. The principal interest, in so far as man is concerned, is centered in the genus *Entameba,* which, among many others, contained the *E. coli* and the *E. histolytica.* The *E. coli* is frequently present in the intestines of healthy people. The *E. histolytica* is probably pathogenic, and may be distinguished by these data: It averages 30 microns in diameter, is grayish and dull-looking, without clearly defined ectoplasm, and possesses a large nucleus. Its movements are sluggish, and when

it becomes encapsulated it divides into eight young entamebæ. The *Entameba histolytica* is a distinct parasite in the intestines of man, and produces there and in other organs most intense destructive changes. It is recognizable by its larger size (20 to 60 microns), and by the striking contrast between its granular cytoplasm and its glassy, refractive, colorless ectoplasm; the latter is usually in active motion, throwing out and retracting pseudopods into which the remainder of the body streams. It multiplies also by fission, and when it undergoes encapsulation divides into four new amebæ. The *Entameba tetragena* is identical with this (McCullum).

It is not likely that the common source of infection is through drinking-water or by vegetables contaminated by feces during the time they were grown. It is more likely that the disease is spread by those ill with the disease and by carriers. The stool habits of Oriental peoples, who wash the anus with the fingers moistened with water which is often kept in a bowl in the toilet and seldom changed, make it more than likely that persons who handle food and water which are soon served at the table may become infected. From a practical standpoint, however, the prophylaxis which has been recommended for many years to boil all drinking-water and abstain from the use of fresh vegetables in a raw state brought about the desired result, although the infection arose from another source. It is quite evident that if vegetables, after having been handled by the fingers of infected servants, are brought to the table without being cooked the disease might be readily transmitted in this way, and also that if servants contaminate the drinking-water shortly before it is served this is another means of conveying the infection. It has been fairly well shown that, in the ordinary environment outside of the human intestine, it is rare for the histolytica to remain alive for more than several hours.

The amebæ, after entering the large intestinal tract, probably reproduce. The young amebæ penetrate the mucosa, probably by passing between the cells of Lieberkuhn's follicles, and then enter the lymphatics and make their way to the muscular coat and the deeper structures of the gut, where they live and feed from the tissue-cells, and, perhaps, the cells of the blood. Often they enter the radicles of the portal vein,

and sometimes the mesenteric arteries, in which event they may cause thrombosis. Those in the portal vein may be carried to the liver and produce abscesses. In the submucosa of the intestines they cause infiltrations resulting in the formation of ulcers, which become infected with bacteria and produce the characteristic ulcers with undermined edges, and with their long axes usually transverse to the direction of the gut. Occasionally an ulcer perforates and peritonitis results. Amebic dysentery may persist for years, and it not infrequently happens that large ulcers are found at autopsy in persons dead from other causes and free from symptoms of dysentery during life. When the ulcers are very numerous, whole sections of the gut may in consequence become gangrenous. Sometimes, after cicatrization takes place, contractions occur which result in stenosis, and, perhaps, in obstinate constipation. The cicatrized ulcers often become pigmented through the interaction of sulphurated hydrogen and blood iron.

As a rule, the cadavers of subjects dead of amebic dysentery are emaciated, and the abdomen is sunken; the *rigor mortis* begins early, is not well marked, and soon passes off, and decomposition begins quickly. On opening the abdomen the coils of the small intestine are usually found to be normal in appearance, but at times they are congested. The large intestine is generally contracted, thickened, and may be gangrenous in sections, or even throughout its entire length. The mesocolic glands are usually enlarged and hyperemic. Commonly the colon is adherent to the liver, spleen, or other neighboring structures. On opening the colon the mucosa will be found red and inflamed, with numerous areas of ulceration and infiltration. These areas are most commonly found in the cecum, the hepatic flexure and the sigmoid colon, but ulcers also occur in other parts of the large intestine. Deeper circular or oval ulcers may be noted with their surfaces covered with the dark-reddish sloughs, their edges undermined, and their bases formed by the muscular coats. Oval ulcers have their long diameters transverse to the bowel, in the majority of instances. Scrapings from these ulcers show blood-cells, leucocytes, bacteria and ameba. The Peyer patches may be enlarged, and the lower end of the small

intestine may show bright-reddish nodules. Frequently an ulcer is found in the vermiform appendix.

The liver is frequently fatty, congested, and may be the seat of one or more abscesses, which vary in size from that of a pea to that of an orange, or even larger. Frequently scar-tissue, the result of former abscesses, is found in the liver. The pancreas is usually normal and the spleen is not enlarged, but at times may contain an abscess. Ordinarily, the kidneys show a parenchymatous inflammation. The contents of the thoracic cavity are usually normal, although at times there may be a hepatopulmonary abscess.

The *onset* of the disease is not always sudden, and at times may be very insidious. The patient may have merely the feeling of an indefinite illness, but, as a rule, in the acute type the onset is abrupt, and is preceded by slight diarrhea, which alternates with constipation; often diarrhea is not present. The stools, which may reach thirty per day, contain blood and mucus, and occasionally greenish matter. On microscopic examination the bowel contents show mucus, amebæ, bacteria, and frequently shreds of tissue. There is usually loss of appetite, and there may be nausea and vomiting, with great derangement of the digestion; the abdomen is retracted, and usually painful upon pressure over the area of the large bowel. Microscopic examination of the blood often shows a reduction in the number of erythrocytes, and there may be a leucocytosis of 20,000 or more per cubic millimeter. Billet states that the number of eosinophiles is increased even in the absence of hookworm or other helminths. The urine is diminished in quantity, and may contain albumin and casts. In the white race the skin often takes on a peculiar pallor. There may be fever of remittent type, but it seldom rises to more than 38.5° C. (101° F.). With the fall of temperature there is usually a decrease in the pain and tenderness in the abdomen. This may be a favorable sign, terminating in recovery, or it may indicate either a gangrenous complication or hemorrhage. If recovery takes place, the stools become less frequent, and are gradually formed, and free of blood and mucus. In fatal cases death often takes place during the first week or ten days from the onset of the acute symptoms. In the chronic type of the disease death often

takes place quite unexpectedly. Persons who are apparently in fair average health apply for hospital treatment, complaining of weakness and exhaustion. They do not give any history of diarrhea or other symptoms to indicate disease of the intestines, and frequently die within a few days after admission; in such instances at autopsy several feet of gangrenous intestine, studded with typical amebic ulcers, near the rectum, is a common finding. Others, subjects of the chronic type of the disease in question, may simply present the clinical picture of an intermittent indigestion, but upon examining the stools *Amebæ histolytica* are found.

Ameba Carriers. It frequently happens that persons are carriers of amebic dysentery who show no sign of illness, nor benefit from specific treatment. At the present time it is not known whether there may be ameba carriers with a normal and intact mucosa of the intestinal tract.

Sometimes it happens that subjects of amebic dysentery also contract the bacillary type of the disease, and in these cases there is generally a higher fever, nausea and vomiting, with marked constitutional disturbance. The end usually comes quickly, and the patient may die in a state of delirium. At rare intervals there is perforation, followed by peritonitis.

The most frequent complication of amebic dysentery is hepatic abscess; other complications, of less frequent incidence, being gangrene of the bowel, peritonitis and excessive hemorrhage. Amebic abscess of the liver usually shows the same symptoms as abscess of the liver from other causes. Tenderness over the organ, with chills and fever, always should be regarded with suspicion.

After a person has apparently recovered from the amebic dysentery a hepatic abscess may occur. Stenoses of the colon, due to contractions of old ulcers, are not infrequent, and are often cause of ill health for many years.

Positive *diagnosis* by clinical means between bacillary dysentery and other forms of diarrhea, accompanied by blood and mucus in the stools, is practically impossible. Diagnosis can usually be made quickly by microscopic examination of the stools and with a view to the demonstration of the *Ameba histolytica* or the *tetragena*.

It is difficult to make a satisfactory prognosis in cases of amebic dysentery, for the disease may have existed for a much longer time than is suspected, and hepatic complications may be imminent. In the acute type Castellani states that hiccough is an unfavorable sign, often indicating the approach of exhaustion and death.

TREATMENT.

Rest in bed is usually of the utmost importance, and this essential precaution is probably more neglected by the patient and the doctor than any other feature of the treatment. Until recent times many drugs have been employed, but upon the advent of the discovery of emetin in the treatment of dysentery by Rogers[81] other remedies have been discarded. The patient should be placed in bed, put on liquid diet, and given a large dose of castor oil. Later sodium sulphate often proves of value. After the purgative has acted, ⅓ grain (0.02 Gm.) emetin hydrochlorid should be given hypodermically; emetin can usually be purchased ready mixed with salt solution in hermetically sealed tubes. Experience of the past few years shows that, while emetin is satisfactory in the majority of cases, still there are others in which it appears to be of no value whatever. Many clinicians believe that this is due to the ameba becoming encysted, and, therefore, uninfluenced by the drug while in that stage. Some success has been obtained by waiting several weeks after the first emetin treatment, in order to give the encysted ameba an opportunity to develop, and thus reach the stage of susceptibility to the effect of the drug. It has also been found that in cases which have resisted the ordinary treatment, active purgation with sodium sulphate, followed by large doses of ipecac, may be of value. Ipecac is perhaps best given in 20-grain (1.3 Gm.) doses, between 8 and 10 P.M., with instruction to the patient to resist, as far as possible, the act of vomiting, which follows the administration of the drug. In using ipecac it is most important to select a potent preparation, and it is generally believed that powdered Brazilian ipecac, with an alkaloidal strength of 2 per cent., gives the best results.

Some success has also followed the treatment of obstinate cases by the use of salvarsan in 10-grain (0.6 Gm.) doses,

repeated three times on different days, if necessary. Still other medical men have combined with emetin quinin irrigations varying in strength from 1 in 5000 to 1 in 750. About 3 pints (1.7 l.) should be injected slowly into an adult by means of a soft rectal tube 32 inches (81.2 cm.) long, and lubricated with carbolated vaselin before insertion. Sometimes the use of the rectal tube can be greatly facilitated by the introduction into the rectum of a 1-grain (0.065 Gm.) suppository of cocain. Many other substances have been recommended for irrigation, but quinin probably has the best record of success. Dr. Shattuck's prescription was very successfully used in Bilibid prison in Manila before the emetin treatment was discovered, and lately the use of this prescription has been resumed in obstinate cases in connection with emetin treatment.

Shattuck's recipe is as follows:

Simaruba pulvis	3.00 Gm. (46.5 gr.).
Benzonaphthol	3.00 Gm. (46.5 gr.).
Bismuth subnitrate	8.00 Gm. (123.4 gr.).
Acacia pulvis	10.00 Gm. (154.3 gr.).
Fluidext. krameria	13.50 mils (3.6 f℥).
Aqua mentha piperit. .....q. s. ad	200.00 mils (6.7 f℥).

M. et ft. mix.

Dose: Tablespoonful (15 mils) every four hours.

For years it has been admitted that the best prophylaxis against amebic dysentery was the use of safe water and the avoidance of eating low-growing garden vegetables in a raw state. Salads were looked upon with suspicion. It was assumed that vegetables were especially dangerous, because they might have been fertilized or irrigated with solutions of human excrement from persons afflicted with amebic dysentery. It was also assumed that pathogenic amebæ lived for long periods of time in many tropical drinking-waters. This is another example of the practice producing the result without necessarily being based upon sound theory. In view of the work of Walker[82] it seems more than likely that infections with amebic dysentery are most commonly transmitted by the means of fingers of persons engaged in handling food. Reference has already been made to the Oriental custom of washing the anus by means of the fingers dipped in water con-

tained in a small bowl immediately after defecation. (See page 308.) Walker showed that it is extremely unlikely that pathogenic amebæ in the stools of infected persons used for fertilizing vegetables remain alive for more than a few days, and probably only for a few hours. Some of the observers have held that the ordinary amebæ are found in practically all surface waters in the tropics and on vegetables, and may, under certain environmental conditions, become pathogenic. If this is the true explanation, the origin of cases of amebic dysentery is much simplified. The scientific world, during the past few years, has been inclined to disregard this view, and large installations of public water supplies and reservoirs have been built without any attempt having been made to remove any of the so-called pathogenic amebæ. Water supplies with amebæ of this kind are used in many cities and districts without any amebic dysentery resulting therefrom. Studies made during recent years in Manila of the stools of persons employed in hotels, restaurants, and other public places where food and drinks are served, have revealed a considerable percentage of amebic dysentery carriers among the servants; and in places where cases of this infection were occurring from time to time, the disease completely disappeared after these carriers were eliminated.

BACTERIAL DYSENTERY.

There is apparently no end to the kinds or the variations of the same bacteria that may be responsible for dysentery. The *Bacillus dysenteriæ* is described as having various strains which take the name of the different persons who have described them, as, for instance, Kruse, Shiga, Flexner, Strong, Hiss, Castellani, Wilmore, and many others. Bacillary dysentery is found in all parts of the world, but it is most common in the tropics. It is probably responsible for a larger death-rate in many countries than cholera or any other disease of the intestines. There is frequently a heavy mortality from infection with the dysentery bacillus in the summer among infants in large cities of the United States and Europe.

BACILLARY DYSENTERY.

Bacillary dysentery is an acute infection provocative of enteritis, and is caused by one of the various types of the dysentery bacillus. It is characterized by diarrhea accompanied by pain, tenesmus, and the passage of blood and mucus in the stools.

With the advent of the discovery of bacteria numerous observers reported different kinds of micro-organisms as being responsible for dysentery and other diarrheal diseases. Probably no other disease in the annals of medicine has had so many distinguished medical authors report upon so many different bacteria as being definitely responsible for its origin. Contrary to many other medical disputes, the controversy, on the whole, has been conducted with admirable good feeling. Considerable light was thrown upon the question between 1898 and 1900 by Shiga,[83] who had returned to Japan after many years' study in Germany. Kruse,[84] at the same period, was making valuable contributions from Germany. These observers described the cause of dysentery as a short bacillus, Gram negative, not clotting milk, and not producing gas in sugar media. The question of motility soon arose, but Kruse's statement that the bacillus was non-motile proved to be correct. Kruse was among the first to call attention to the fact that there was more than one variety of the dysentery bacillus. After him came a long list of observers, who from dysenteric stools isolated bacilli which varied in some of the details from those described by Kruse and Shiga.[85] Flexner[86] reported a dysentery bacillus of moderate motility in Manila. Strong[87] isolated another bacillus in Manila slightly different from that of Flexner. In 1903 Hiss and Russel[88] described a bacillus, frequently referred to as the *Y*-bacillus, which resembled Flexner's, but failed to ferment saccharose. In 1904 Castellani[89] isolated another bacillus in Ceylon; various observers in the United States found bacilli resembling the Kruse type from cases of diarrhea in children. In brief, the literature fairly teems with observations upon bacteria that are supposed to be associated with dysentery; and, owing to those discrepancies, the whole question, from the scientific standpoint, is still in a very unsatisfactory state.

Bacillary dysentery is found in all latitudes, and should be regarded as a great tropical scourge. In the tropics it is probably responsible for a greater number of deaths and cases of illness than any other disease. Cholera is frequently much more spectacular, but a careful study of the health conditions in any tropical country will usually show that bacillary dysentery is responsible for a greater morbidity and mortality. The eating habits of the people, especially Asiatics, and the temperature favorable for its spread are operative throughout the whole year, premises that make the disease a formidable opponent of the sanitarian. In countries like New Guinea, Java, Ceylon, India, the Philippines, the victims of dysentery die literally by the thousands, but, as the mortality does not approach that of cholera, it attracts less attention. The deaths, especially among children during the summertime in the larger cities throughout the world, also is a very important factor in the mortality figure. It has been noticed in the Philippines, for example, that the disease will break out simultaneously on many different islands, and careful inquiries made over a period of years have failed to show any connection between cases occurring on different islands, although, after the disease once makes its appearance, its spread can frequently be traced from place to place. Very often the affection makes its appearance at the head waters of rivers, and spreads to the towns and villages which lie along their banks. It has been frequently observed that the spread of the disease is intimately connected with the presence of flies. Again and again outbreaks of bacillary dysentery have been stopped when human excrement was collected in fly-proof receptacles, and measures taken to eliminate flies and to prevent their gaining access to human stools. The disease is often associated with large collections of people among whom provision for the safe disposal of human excrement is lacking; thus, it is frequently associated with armies in time of war, and with religious pilgrimages, camping parties, etc. Many observers also believe that dark, damp days greatly favor the spread of the disease, in that the organism has a longer life under favorable weather conditions, and thus has many additional opportunities to find its way to the intestines of human beings. Great epidemics of the disease have

occurred in the past, and spread over whole countries. Noteworthy epidemics occurred in Europe at numerous periods, the last recorded being that of 1834 to 1836. Smaller epidemics occurred very frequently in institutions like orphan asylums, insane hospitals and prisons.

Bacillary dysentery is usually ascribed to bacteria which belong or are allied to the principal types. Type one is the Kruse-Shiga *Bacillus dysenteriæ,* and others are sometimes described as variants. The bacilli are found in the dejecta, and can be recovered in scrapings taken from the surface of the bowel *postmortem.* The literature of the various kinds of bacteria associated with dysentery is most extensive, but a review of it does not seem to be indicated here. The disease is spread by the feces of persons suffering from the infection, and also by persons who are merely its carriers. Strong and Musgrave[90] have reported a case of infection in a man to whom they gave a pure culture of the dysentery bacillus, which soon provoked an attack characteristic of dysentery. The disease is probably spread in the same manner as typhoid fever, and can be contracted only by conveying to the mouth parts of stools which contained dysentery bacilli. There are many ways in which this may be brought about, although absolute and definite proof covering any considerable number of cases is lacking. The most probable methods are, perhaps, by the fingers coming in contact with dysentery stools and then infecting the food or drink of other persons. Another common method of infection is by flies which have access to dysentery stools, and later contaminate food and drink. It has been asserted that dried dysenteric stools, which may be blown about in the form of dust, may be responsible for the dissemination of the disease. It has been demonstrated many times that flies may have dysentery organisms on their feet, and that even the excreta of flies, which have fed on dysenteric stools, may contain the bacilli; and in this way transfer them to food and drink. The dysentery bacilli have been found in the feces of monkeys and rabbits, and it would appear that animals might be a factor in the spread of the disease. Many outbreaks in camps, public institutions, and even private houses, have been traced to persons who were employed about the kitchen in preparing food. A theory also

has been advanced that the bacilli are capable of living in the alimentary canal without causing symptoms until the vitality of the host is lowered by chill, indigestion, or some other intercurrent affection. This might explain the simultaneous appearance of the disease among persons residing over large areas of territory, and even on different islands, and without any apparent communication between them, but at best the explanation is very unsatisfactory. The dysentery carrier is probably a most important factor in the spread of the disease. Observations made during a dysenteric outbreak have shown that a considerable percentage of apparently healthy individuals may harbor the specific bacilli. Persons who have at some previous time had an attack of dysentery are regarded with great suspicion, and examinations made of their stools many months after the attack have shown the presence of the dysentery bacillus.

Dysentery bacilli taken into the body with food and drink apparently do not multiply until after they have passed the stomach. They may be found growing throughout the whole length of the intestines, the colon being principally involved. The micro-organism produces toxins absorbed into the blood, of which two are known. One acts upon the large intestine, and the other on the nervous system. One is excreted by the large intestine, and causes the lesions which are associated with dysentery. In the process of excretion the tissue of the muscular and mucous coats is destroyed by coagulative necrosis and thrombosis. This process creates the diphtheroid membrane which is first noted on the summits of the ridges, and later spreads to the space between the ridges. Micro-organisms destroy this diphtheroid membrane, which then separates off in flakes, leaving ulcers which are at first superficial, but later become deep. These ulcers heal with the formation of scar-tissue. The toxin of the disease may attack the nervous system, and cause peripheral neuritis. At times the bacilli enter the blood and cause septicemia. Darling[91] has reported cultivation of the bacilli from the blood of cases of bacterial dysentery. Painful joint changes, causing effusion into the joints, are not uncommon, the ankles, knee and hip being most frequently involved; it often happens that when one joint clears another becomes affected.

Usually the peritoneum is normal, but the blood-vessels of the colon are injected, and the mesocolon may be infiltrated with lymph; often there are adhesions of the sigmoid colon to the omentum, pelvis, bladder, or small intestine—in fact, adhesions are often general. The quantity of peritoneal fluid is usually normal, and the bowels do not have the sticky condition so frequently found in cholera. The small intestine may be hyperemic, but this does not differ from the condition found in many other diseases; at times there are ecchymotic spots on the walls of the gut. The large bowel may be gangrenous along varying lengths of its extent, and on opening it the mucous surface is covered with a coagulated exudate in the form of a false membrane, the surrounding areas of the mucosa being hyperemic and edematous. As a rule, there are many ulcers with clean surfaces and elevated edges, and this ulcerative process may be very extensive. Other spots of ulceration are covered with sloughs, which may extend deeply into the bowel and cause perforation and peritonitis. In cases which have died from some other complaint the intestines are often found matted together. In the colon will be found many scars with pigmented deposits indicating the site of former ulcers, and sometimes scar-tissue will have contracted and reduced the lumen of the bowel to small proportions. The cecum may contain polypi, which protrude from the mucous membrane, and this state of affairs is ordinarily referred to as colitis polyposa. In children there is often a hyperplasia of solitary gland-follicles. Microscopically, the exudate at first contains few cells and the mucosa is seen to be congested; later the exudate into the submucosa seems to change into fibrin and the vessels are dilated, and contain numerous polymorphonuclear leucocytes extravasated into the surrounding tissue.

Bacillary dysentery is frequently described as occurring in distinct types, but in actual practice it is most difficult to differentiate between them. The *symptoms* largely depend upon the severity of the infection, and the resistance of the individual. There are, however, outbreaks in which the vast majority of the cases are of an acute mild type with a low mortality. In some individuals, in whom cicatricial changes in the bowel have taken place, the disease may become

chronic, and last over a long period. In fact, some subjects suffer from chronic dysenteries or its effects for years after the acute symptoms have subsided. During some outbreaks the gangrenous type predominates. Again, there are outbreaks in which children are mostly affected.

In acute dysentery the *incubation period* varies from one to three days. It usually begins with loss of appetite and malaise, followed by pain in the lower abdomen, and an urgent desire to defecate. The first movements are usually formed stools, and some relief is obtained afterward, but this is usually followed by another attack of pain, and the stools become more and more liquid, until finally blood, flakes and mucus is passed. At the height of the outbreak, practically only blood and mucus are passed. The lower bowel becomes raw and the anus inflamed and painful, and prolapse of the bowel may occur. The urine becomes scanty in amount, causing an apparent increase in the quantity of urea. In mild cases there is a bowel movement about once an hour, but in severe cases there may be fifty or more during twenty-four hours. The constant defecation prevents sleep and rest, and the patient becomes greatly exhausted from physical exertion alone, which, added to the toxemia produced by the disease, weakens the patient very rapidly. In spite of the constant diarrhea, there is no great thirst, and this craving is usually satisfied by small quantities of water. Nausea frequently occurs, and there is striking anorexia with digestive disturbances. The whole abdomen becomes painful and sore, and, in untreated cases, vomiting is frequent. It often happens that formed feces accumulate in the upper part of the intestines, which are not affected by the disease. Typical dysenteric stools are composed of blood and mucus only, but in more serious cases there are numerous white shreds as well. Microscopically, numerous bacteria can be seen, leucocytes, erythrocytes and epithelial *débris*. The temperature ranges from 38.6° to 39.5° C. (101° to 103° F.). The pulse is accelerated, and in serious cases it may be irregular. No marked changes occur in the blood, although there may be an increase of the polymorphonuclear leucocytes. The lungs are normal and delirium is unusual. In serious cases the foregoing symptoms increase in severity; the temperature drops suddenly to

normal; the motions decrease in number; hiccough appears, and, after a stage of exhaustion, death occurs, commonly during the second or third week of the illness. If the patient is to recover, the symptoms gradually improve, blood and mucus disappear from the stools, the tongue becomes clean, and convalescence usually begins about the end of one week. In severe cases convalescence may be prolonged for as long as a month.

Gangrenous Dysentery. This type of the disease, as a rule, begins very insidiously, and may not attract attention until a few hours before death. The patient suddenly collapses, and dies within a few hours. The typical dysenteric stools are not present in this form of the disease, but in most cases the onset occurs during an attack of acute dysentery, all of the symptoms of which become greatly aggravated, and the stools become very offensive and contain gangrenous sloughs, which are composed of the lining membrane of the intestines. At times these sloughs are sufficiently large as to appear in the form of cylindrical casts of the intestine. Recovery from this form of dysentery is extremely rare.

Enterodysentery. This form usually begins with chills, and the temperature rises to about 103° F. (39.5° C.), tending to assume the continuous type; the tongue is dry; the mouth is covered with sordes; the breath fetid, and there is headache, with pains in various parts of the body, and often ecchymoses under the skin. The patient is usually stupid and at times delirious; later abscesses frequently appear, especially in the parotid or the ischiorectal fossæ. Carbuncles, bed-sores, and peritonitis also frequently occur. Death usually takes place before the end of the first week.

Chronic Bacterial Dysentery. This type of the disease usually follows the subsidence of an attack of the acute form from which the patient apparently has recovered. There are usually five or six daily bowel movements, which are evil-smelling, and sometimes contain blood and mucus. Blood, however, is often frequently absent. This is followed by alternate periods of diarrhea and constipation. The tongue is unusually red and clean. Digestion is impaired, and fermentative changes are frequent. Profuse night-sweats are com-

mon. Recovery often takes place spontaneously. Patients frequently die of some intercurrent disease.

Infantile Diarrhea. This is an extremely common disease among infants in the tropics, and in nearly all respects is similar to the dysentery or diarrhea outbreaks which occur during the hot months in American cities. There is considerable difference of opinion as to whether the Flexner or the Kruse-Shiga type of bacillus predominates. The symptoms are similar to those described under Acute Dysentery. The onset of the disease is usually ushered in with vomiting and a rise of temperature from 103° to 104° F. (39.5° to 40° C.), the tongue is coated, the abdomen extended and tender, and at first the stools are green in color, but later they are mixed with blood and mucus. The fever is often of the remittent type. Death may not occur for a number of weeks. The child usually wastes, and often after repeated attacks of diarrhea it finally succumbs to exhaustion. In more favorable cases it often requires months to recover from the attack.

Bacillary dysentery must be carefully distinguished from amebic dysentery, and the other many forms of diarrhea which are so common in the tropics. The typical bloody stools, the absence of the *Ameba histolytica,* and the presence of one of the dysentery type of bacilli usually make the diagnosis reasonably easy. As a rule, the disease also occurs in epidemic form, and it is more prone to be widespread than limited to a few cases. Care must, of course, be exercised to exclude the presence of blood in the stools from hemorrhoids, cancer, syphilis, or similar cause.

Peripheral neuritis occurs as a complication in a fair percentage of cases. Peritonitis is not uncommon, as well as inflammation of the tendon sheaths. In gangrenous dysentery hemorrhage may be a serious complication. Typhoid fever and dysentery may occur in the same time, and cases of appendicitis have often been known to occur.

Probably the most serious complication is the stenosis due to scars of the intestine left by the healing of the ulcerated surfaces. The most constant sequela is probably constipation, with the train of symptoms which follow in its wake.

A fair average mortality for dysentery is probably 15 per cent. The site of the disease is an important factor; if

located low down in the bowel, the prognosis is much better than when situated higher up. Apparently persons who have had the disease before, or who have resided for many years in places where the disease prevails, are less susceptible to an attack, and do not suffer as severely as newcomers. It has recently been observed in a jail at Borneo that prisoners from the lowlands show a much lower infection rate in the jail than prisoners from mountain districts. It is very well recognized in Borneo that dysentery prevails very extensively in lowlands, and only seldom in high elevations.

TREATMENT.

Much has been written upon the treatment of bacillary dysentery, and many remedies are suggested, but a review of the field shows there is no great difference between the results, regardless of the remedy employed. This warrants the inference that the disease is not much influenced by the various methods of therapy which have been advocated. Probably as good results as any are obtained by *rest in bed,* liquid diet without milk, and such symptomatic treatment as may be necessary in order to make the patient comfortable. It is well to begin the treatment with a suitable dose of castor oil; this is to be followed with 5 grains (0.325 Gm.) of bismuth salicylate and 5 grains (0.325 Gm.) of salol, made up in cachets and administered every two to four hours. The frequency of the dose should be reduced as the stools become less. Large enemata of warm normal salt solution, of borax, or of bicarbonate of soda in the strength of 5 grains (0.325 Gm.) to the ounce (30 mils), often give much relief. This should be given with a long rectal tube, well lubricated before it is introduced. If pain and discomfort continue, it should be controlled with hypodermic injections of morphin. In spite of the many favorable reports which have been made upon the use of the different serums, the fact remains that some thousands of cases treated without serum show no great difference in results. If serum is to be used, it is better to use one of the polyvalent preparations. Astringent drugs like tannin and bismuth subnitrate are often found useful in relieving the symptoms. Collapse should be combated by hypodermic or intravenous injections of normal salt solution.

The use of calomel, after the administration of the castor oil, is a favorite with many doctors, and ½ grain (0.03 Gm.) of mercury salt is given hourly until twelve doses have been taken; this regimen is usually repeated for three days. The saline treatment is another favorite. Castellani[92] recommends the following: Two drams each (7.80 Gms.) of magnesium sulphate and of sodium sulphate, dissolved in an ounce (30 mils) of water, should be administered, and then 1 dram (3.75 mils) of each is given hourly, or every two hours, until the motions become feculent, and then every three or four hours for another day. The sour-milk treatment is advocated by many, especially for subacute and chronic cases. In chronic cases the constipation is, perhaps, best relieved by liquid paraffin, but if there is actual constriction, the necessary surgical cure should be immediately sought.

Success in the prophylaxis of dysentery consists of preventing the stools of the persons who have or have had the disease, or of carriers reaching the intestinal tracts of non-infected persons. The following rules to prevent this have long been issued by the Health Department of the Philippines, and have been found to be very effective in practice.

These diseases can be introduced into the system only through the mouth. They are caused by organisms too minute to be seen, except with a microscope, but which may be readily killed by heat, as well as by disinfectants, thus making it possible to combat such diseases successfully by the use of fire and boiling water when there are no chemical disinfectants available.

The following precautions should be taken at all times:

1. Use only boiled, distilled, or bottled water, or water from an approved artesian well, for drinking purposes or for cleansing the teeth and mouth.

2. Always wash the hands thoroughly after coming from stool and before eating, and see that the servants do the same. In times of epidemics the use of a 1 per cent. solution of tincture of iodin or a 1:2000 bichlorid solution for submerging the hands after washing them affords additional safety.

3. Do not touch water or food with the hands unless they have just been washed, well dried, and disinfected when prac-

ticable. These precautions must be enforced on the servants, since it is often by their carelessness that such diseases are spread.

4. All food should be cooked. Fruit that grows on trees well above the ground may be safely eaten, unless it has been contaminated by handling.

5. Flies may carry the organisms of dysentery, cholera and typhoid on their feet; therefore, as a protection against contamination from this source, all food should be covered as soon as it is cooked.

6. All manure and garbage should be kept in covered receptacles, and properly disposed of to prevent the breeding of flies.

7. Boil all water used for diluting milk.

8. Keep kitchen and table dishes thoroughly clean, and scald the dishes each time before they are used.

9. Vegetables and fruit which grow on or near the ground should not be eaten unless cooked. Raw vegetables are dangerous.

10. No diarrhea or disorder of the bowels, however slight, should go untreated.

11. The bowels and other eliminating organs of the body should be kept in good condition.

12. The dejecta of dysentery patients, as well as that from typhoid and cholera patients, should be thoroughly disinfected by adding to them two or three times their bulk of 5 per cent. carbolic acid, a 1 : 1000 solution of formaldehyd, a 5 per cent. solution of creolin, a 1 : 500 solution of larvacide, or by burning or boiling them. The disinfectant and the stools should remain in contact for at least one-half hour, and then they may be disposed of in the closet, or by burying and covering with earth.

RAT-BITE FEVER.

Rat-bite fever is an infectious disease following the bite of a rat, and includes similar fevers which at rare intervals are consequent to bites of weasels, ferrets and cats.

Rat-bite fever is referred to frequently in ancient Japanese books. One of the first fairly complete reports is that of

Miyake made in 1899, when he reported 11 cases.[93] Other cases have since been recorded by Horder in England,[94] by Proescher, Blake, and Tileston,[95] in the United States. Recently many cases have been reported from India, and the disease has been under investigation in that country for some time.

The religious belief of many of the Indians, which prevents them from killing animals of all kinds, has resulted in an enormous rat population. As rats are not nearly so shy in India as in other countries, consequently they live in closer relationship to man, and the opportunities for the spread of the infection are greater.

Recently Tileston has reported 2 cases from New Haven, Connecticut.[96] Previously Blake had reported the presence of the disease in Boston, Massachusetts.[97] There is much reason to believe that rat-bite fever is much more common in the United States than has heretofore been thought probable.

In 1914 Schottmüller described a streptothrix, which he named the *Muris ratti*.[98] He found the micro-organism in eight successive blood cultures in a case clinically diagnosed as rat-bite fever. More recently Blake has isolated in a fatal case of a rat-bite fever a micro-organism practically identical with that of Schottmüller.[99] He obtained the germ in pure culture from blood during life, and from the heart's blood *post-mortem*, and also from a diseased mitral valve. Futaki[100] and others have reported the finding of spirochetes in the skin and lymph-nodes, with successful inoculations into animals. There is, therefore, no agreement as to the true causative agent.

The chief characteristics of the micro-organism found by Schottmüller, Blake and Tileston are the following: It is a branching, filamentous germ varying greatly in length, and showing a tendency to fragment into smaller forms resembling both bacilli and cocci. They stain with the usual stains, and are negative or faintly positive to Gram's stain. They are only found in fresh blood-smears taken during the febrile periods.

The *incubation period* of rat-bite fever varies from one to sixty days, with an average of two weeks. The onset of the disease is characterized by a febrile attack with a step-like

rise which, by the end of the third day, frequently reaches from 104° to 105° F. (40° to 40.5° C.). There is then a fall by crisis, after which the temperature remains normal for a few days, and there is then a repetition of the attack described above. The febrile attacks may continue over a period of many weeks. Cases have been reported in which attacks are repeated at intervals of months, or even years. Hora reports a case in which there were ten relapses.

There is also another type of the disease known as the Abortive Type. The fever is not high, and local symptoms are prominent. Sometimes there is a continued fever, in which there are marked symptoms of the nervous system.

There is nearly always a localized bluish-red erythema with sharply marked outlines covering an area of a few inches around the wound produced by the bite. There may be a generalized eruption consisting of bluish-red spots, circular in form, 0.5 to 3 cm. in size. They are usually first noted over the lymph-nodes draining the region of the bite. Later it may spread over the body, showing no special predilection for any particular surfaces. In the course of a few days these spots usually become ring-shaped, due to fading of the centers.

Soon after the bite of the rat the site of the wound becomes red and swollen. An ulcer frequently forms, and the regional lymph-glands become enlarged. The enlargement of the glands is usually coincident with the onset of the fever, which may be accompanied by a sensation of chilliness.

The characteristic temperature curve following the bite of a rat, or that of a ferret, weasel, or cat which has come closely in contact with rats, is an important diagnostic feature. The fever is distinguished from ordinary wound infection by the length of the incubation period. In rat-bite fever it is usually several weeks, whereas in an ordinary wound infection it is only a few hours. The local appearance of the two types of wounds may be similar. Abscess formation is rare, although occasionally there may be necrosis and grangrene at the seat of the bite. An important distinguishing feature is a localized redness above the joint nearest the site of the wound. The generalized form of the eruption, when typical, may be regarded as pathognomonic. It consists of fairly numerous,

large, bluish spots, which are nearly circular in shape and slightly raised, with sharply defined margins. The spots vary in size from a few millimeters to 4 centimeters (1.3 in.). A few days after their appearance they frequently become ring-shaped, and resemble the lesions of erythema multiforme. The spots disappear on pressure; do not itch or scale. The finding of the organism in fresh blood-smears may be regarded as fairly conclusive.

The mortality in the past has been only about 10 per cent. Death is most usually due to blood-poisoning during the first attack.

TREATMENT.

The rat-bite should be immediately cauterized, either with pure carbolic acid or with the actual cautery. When done within an hour it is said to be an effective preventive.

Public-health measures should consist of destruction of house-rats and the enforcement of regulations to bring about rat-proof construction in new houses, and, at least, palliative structural changes in human habitations already built.

In 1912 Hata[101] reported 8 cases treated with salvarsan, of which 5 were cured by the first injection. Of the remaining 3, 1 had a single relapse, which subsided without further treatment. The second was not followed further, and the third, a 2½-year-old child, was not cured after the third injection. Similar good results with salvarsan have been reported in India and America. Probably the best results are obtained by the administration of 0.3 gram (4.6 grs.) of salvarsan intravenously.

RELAPSING FEVER.

Relapsing fever is one of a group of dangerous communicable diseases, characterized by fever of sudden onset, with rapid lysis after the seventh day, with relapses varying from one to seven days in their frequency. The disease is caused by a spirillum which is constantly present in the blood during the febrile periods.

It is a disease that is extensively distributed throughout the tropical and temperate zones. Unlike most other diseases, the type of organism frequently varies in the different coun-

tries. For instance, in Europe the spirillum is of the type described by Schaudin; in the United States, that of Novy; in West Africa, that of Dutton; in India, that of Carter. The micro-organisms in the different countries vary somewhat in their morphology and cultural characteristics. Relapsing fever also differs from other diseases, in that it seems to be transmitted by different insects. For instance, it seems possible that it may be transmitted by ordinary ticks, bedbugs, fleas, biting flies and lice. It is also shown by Leishman[102] that the *Spirocheta duttoni* may be transmitted hereditarily in the tick. Positive results have been obtained in the second generation, the bites of which were infective for mice and monkeys. Third generation infections among ticks, so far, have failed.

Febris recurrens, spirillum fever, five-day fever, typhus recurrens, icteric typhus, remittent fever, bilious typhoid, and epidemic remittent fever are synonymous names for this infection.

Relapsing fever has figured in medical literature since the very earliest times. It was known to Hippocrates, who described an epidemic in Thasos. However, there are no further references contained in medical literature until 1770, when it was described by Rutty as a disease common in Ireland. Obermeyer observed a spirochete in the blood in 1868, but did not publish the result of his observations until 1873 after having had an opportunity to witness a subsequent outbreak and verify his observation. The micro-organism was named *S. obermeieri* by Cohn in 1875. In 1874 it was named *S. recurrentis* by Lebert. Obermeyer's observations were confirmed by Münch, of Moscow, when he inoculated blood from a person sick with the disease into a healthy human being, with positive results. Metchnikoff later made successful inoculations into mice and monkeys. In 1904, Philip, Ross and Milne, and still later, Dutton and Todd, discovered in Africa that the spirochete was communicated by the bite of the tick *Ornithodorus moubata.*

The disease occurs in Great Britain, especially in Ireland, Norway, Denmark, Germany, Russia and Turkey. Severe outbreaks and many epidemics of the disease have taken place in Russia. It also occurs in Egypt, the Sudan, Algeria, Congo Free State, Angola and German East Africa. It is very com-

mon in China, and cases have been reported from Hong Kong. It is also common in Sumatra and India. Its last known appearance in the United States was in New York and Philadelphia, in 1869. Some textbooks state that the disease occurs in the Philippine Islands, but it has not been reliably reported from that country. It is also suspected to exist extensively in Central and South America, but reliable data are lacking.

Animal experiments have shown that at least one form of relapsing fever is caused by the *S. recurrentis,* which is a delicate, spiral filament, from 7 to 9 microns in length and 25 microns in breadth, with a long flagellum, which adds an additional 5 to 7 microns to its length. The parasite usually has from 3 to 6 spirals. In fresh blood preparations the micro-organism is propelled by the flagellum, and shows active screw-like movements. Occasionally very long flagella are seen, and lengths up to 100 microns have been reported. The disease is probably transmitted by some blood-sucking insect. The bedbug has been frequently implicated. Ticks are also, in all probability, responsible for its transmission, as well as a number of other insects. It may also be transmitted by direct inoculations. Accidental infections in laboratories have been frequent.

The life cycle of the *S. recurrentis* is not definitely known. Fever occurs during the time the spirochetes are in the blood, and when they disappear from the blood the temperature falls to normal. It is not known definitely whether the fever is due to a toxin. Immune bodies are supposed to kill most of the spirochetes, but a certain number remain resistant, and recurring attacks of fever take place. A temporary immunity, which may last for several months, is usually acquired. Serum from immunized animals shows definite protective and curative properties.

Necropsy reveals an enlargement of the liver and spleen. The latter organ sometimes attains huge size, and extends well down to the pubes. On section the spleen is dark-colored, soft, with enlarged follicles with congestion and cellular increase. The liver is enlarged, the lobules poorly defined, and cloudy swelling occurs. The kidneys are enlarged and congested, with cloudy swelling and fatty degeneration. The lungs often are hypostatic. The bronchi are generally con-

gested. There is usually a marked polymorphonuclear leucocytosis.

The *incubation period* is said to vary between two and twelve days, and is believed never to be longer than fourteen days. The onset is usually sudden, although in a small percentage of cases it is gradual, with rheumatic-like pains, headache and constipation. When the disease has a sudden onset there is usually a severe chill, pains in the back and limbs, with epigastric pain and tenderness associated with considerable debility. The face becomes flushed, the conjunctiva injected, and the temperature may rise to 104° F. (40° C.) with a proportionate pulse rate up to 112. The fever remains nearly stationary until the sixth or seventh day, when it falls by rapid lysis. The skin is yellowish in color, hot, and usually damp from perspiration. Frequently there is a rose-colored macular eruption, which disappears on pressure upon the thorax, abdomen and legs. The tongue has good tone, is pointed, red at the tip, and the remainder heavily covered with white fur. There is usually constipation. The liver is enlarged and tender, as well as the spleen. There are no changes in the heart. The pulse is that of ordinary fever. There is reduction in the number of erythrocytes and in the percentage of hemoglobin, with a polymorphonuclear leucocytosis. Spirochetes may be seen in the blood, and occasionally are inclosed in a leucocyte. A cough and other bronchial symptoms are common. The crisis is usually preceded by a severe chill, after which there is violent perspiration or diarrhea, often with epistaxis. The temperature falls rapidly during the crisis, and the pulse and respirations assume normal rhythm. The patient usually falls into a sleep, and awakens feeling much refreshed. A remission then occurs, during which the abdominal organs resume their natural size, and the patient's strength gradually returns. Usually about the fourteenth day from the onset a relapse occurs with all the symptoms of the original attack. Second relapses are rare.

The lungs and bronchi are frequently affected. Often there is dysentery, diarrhea and hematemesis. In pregnant women abortion often takes place.

The mortality is usually below 6 per cent., but it may vary considerably in different outbreaks and in different coun-

tries. For instance, in Egypt outbreaks with a mortality of over 14 per cent. have been reported. In the feeble and the senile, death often takes place at the height of the first paroxysm.

The temperature chart in relapsing fever is characteristic, and usually is of great assistance in making a differential diagnosis. It may be distinguished from malaria, typhus, typhoid, and yellow fever, by the presence of the spirochetes in the blood. Agglutination may also be depended upon.

TREATMENT.

Salvarsan acts almost as a specific. Persons who have been debilitated with long residence in the tropics do not seem to bear the administration of salvarsan well, and great care should be exercised. It is better to begin with a small dose, 0.3 or 0.4 gram (5 or 6 grs.) of salvarsan, and gradually increase the dose. Death has occurred even when small doses of salvarsan have been used. In view of the specific effect of salvarsan, the use of other drugs has been largely discontinued. Symptomatic treatment should be carried out. Ice-bags on the head for the headache, or the administration of small doses of caffein and acetanilid, are indicated. The epigastric pain may be relieved by fomentations sprinkled with tincture of opium. Vomiting is sometimes treated with considerable success with bismuth or iced champagne. Effervescing carbonate mixtures are often very useful. In brief, in addition to the salvarsan, the remaining treatment and nursing should be the same as in simple fever.

The prevention of the disease is based upon personal and domestic cleanliness, and avoidance of ticks and other bugs. A mosquito bed-net, in a thorough state of repair and reliably tucked in, invariably should be used.

FRAMBESIA.

Commonly known as yaws, in Spanish-speaking countries, for instance, in the Philippines and South America, frambesia is frequently referred to as bubas, and in the French colonies as pian. Continental authors generally use the term frambesia. The latter term was first used by Sauvage in 1750, on

account of the raspberry-like appearance of the eruption. Tropical polypapilloma, Castellani's spirochetosis, parangi (Ceylon), buena (Burma), puru (Borneo), patek (Netherlands Indies), coco (Fiji), and tona (Tonga Islands) also are used synonymously.

Yaws is a specific infection caused by a treponema, and manifests itself in the early stages by cauliflower-like excrescences of the skin, and later by many manifestations resembling syphilis.



Fig. 13.—Typical case of yaws, Polynesian child.

The thirteenth chapter of Leviticus is ordinarily interpreted to refer to leprosy. It has been suggested by Hume, Adams, and others, that the Israelites were, in reality, afflicted with yaws. Apparently the first descriptions of the disease made by European physicians were based on American experiences. These descriptions refer generally to a disease called pyans or yaya among the natives of the West Indian Islands. In 1718 Bontius described frambesia as being endemic in the West Indies, Java, Sumatra and other islands of the Eastern Hemisphere. It is stated that outbreaks of frambesia frequently occurred among African slaves en route to America, and that it was customary to build hospitals for the treatment

of yaws upon the arrival of these slaves in the West Indies. By some writers mal de chicot in Canada and radesyge in Norway and Sweden are considered to have been yaws. In the Philippines, for instance, the disease referred to nowadays as yaws very often includes many skin affections which are not yaws. It is more than likely that the descriptions of olden-times contained similar errors. From very early times authors have considered frambesia to be a form of syphilis. In 1882 Charlouis endeavored to prove that syphilis and yaws were two different diseases. Probably one of the most classic reports on the disease is that by Numa Rat, published in 1891. In recent years the disease has been very extensively investigated. Probably the most exhaustive work done is that of Castellani. In some countries, in which yaws prevails, it is customary for mothers directly to expose their children to those having the disease. The child that has not had an attack of yaws is looked upon as being deficient. This practice is especially common in Fiji and Samoa.

Yaws is found in most tropical countries. It is especially common in the Philippine Islands, Ceylon, tropical Africa, Fiji, Samoa, and other islands of the South Pacific. It also prevails very extensively throughout the West Indies. Yaws is seldom seen in China, and with few exceptions occurs only in South China through direct importation.

From time to time many different micro-organisms, especially bacteria, have been described as the specific cause of yaws. For instance, Eijkman found peculiar bacilli. Pariez described a micrococcus. Powell cultivated a yeast micro-organism. Nicholls and Watts isolated a coccus, which, however, failed to produce the disease when inoculated into animals. In 1905 Castellani observed a treponema, or spirillum, which he named *Treponema pertenue*. This organism is very generally accepted as the true causative agent. It is constantly present in the primary lesion, and in the unbroken papules. It may also be found in the spleen, lymphatic glands, bone-marrow, and other organs. Up to the present time it has not been possible to demonstrate the germ in the blood, but blood inoculations result positively. In tertiary lesions treponema have not been satisfactorily demonstrated. Positive inoculation experiments are recorded as early as 1848. According to

Charlouis and Castellani, it is possible successfully to inoculate syphilitic patients with yaws, and yaw patients with syphilis. It would seem, however, that considerably more work is desirable before general acceptance of this statement can be expected. In the Fiji Islands, for example, yaws prevails very extensively among the Fijians, and no cases of syphilis have been recorded. Yet it seems almost unbelievable that the Fijians, who have been in intimate contact with Europeans and Tamils, among whom syphilis exists, should not have been exposed to the infection of syphilis. Inoculation experiments in the higher monkeys result positively. In genus macacus yaw lesions apparently only occur at the site of the inoculation, whereas in the ourang-outang lesions similar to those in humans result from experimental inoculations. The disease is apparently neither hereditary nor congenital. The exact method of its transmission is not known, although it is believed to be by direct contact. Infection, however, is said not to occur unless there has been some solution of continuity in the skin. Insect transmission has been suspected, but not proved. No age is exempt, although in endemic countries cases in persons over 12 years of age seldom occur. It is presumed that most of them have had the disease in childhood. One attack apparently confers immunity. The apparent freedom of the Fijians from syphilis would lead to the inference that perhaps an attack of yaws confers immunity to syphilis, much in the same way as an attack of cow-pox protects against smallpox.

The *Treponema pertenue* is slender and spiral-shaped, and varies in length from 18 to 20 microns. Some difference exists in the diameter of the spirillum. It is difficult to stain, although fairly good results may be obtained with the Giemsa or the Leishman method. The ends of the parasite are often pointed, although this is not constant. At times there is a bulbous expansion of an end, which may, perhaps, be due to folding. There are from six to twenty coils. These may be more or less evenly distributed, or they may be concentrated in one small section of the spirillum. The treponema of yaws resembles that of syphilis very closely. The distinction is at present not definite, and the differentiation depends largely on the ability and experience of the laboratory worker. Inoc-

ulation experiments indicate that the spirillum of yaws and that of syphilis are two distinct entities, as they apparently produce totally different lesions. Castellani, in 1911, showed me an apparent case of syphilis in a boy whom he had successfully inoculated with yaws, thus affording additional evidence that they are distinct. The facts previously mentioned with regard to the absence of syphilis among Fijians are, however, very difficult to reconcile with the Ceylon experience. Various forms of bacteria may be found in the open lesions of yaws, but these are not believed to have any special pathogenic significance.

There are no reliable data available to prove that yaws primarily causes death, but the disease has been observed in persons dying of other diseases. In the more advanced cases of yaws, ordinarily described as the tertiary stage, lesions similar to those of syphilis may be found. Gummata, however, have not been reported. Bone lesions are exceedingly common, especially those accompanied by atrophy and absorption. Extensive ulceration with sinus formation, especially of the feet, is very common in untreated cases. The foot may be huge in size and resemble a mycetoma. Ulceration and necrosis of the bones, particularly those of the head, producing scars similar to those seen in syphilis, are also common. Scars on the frontal bone are frequently seen. Periostitis, especially of the anterior tibial surfaces, is common.

There is considerable difference of opinion as to the true primary lesion in yaws. Many observers regard the so-called "mother yaw" as the primary lesion. Others regard yaws as occurring in three stages, and believe that the characteristic skin eruption of yaws is a secondary manifestation. It may be stated, however, that no satisfactory description has been given of an initial lesion other than that of the primary yaw. The exact mechanism by which infection is transmitted to the human being has not been satisfactorily proved. It is assumed to be a contact infection. Certain it is that successful inoculation can be made by rubbing material from a yaw lesion on the skin of a child who has heretofore not been afflicted with the disease. Mosquito and insect transmission have also been suspected, but no satisfactory proof exists. Perivascular mononuclear infiltration and endothelial proliferation in the

walls of the blood-vessels, which are so characteristic of syphilis, have not been reported in frambesia. The typical skin lesion or yaw is a granuloma. After it has reached a certain stage there are evidences of hyperkeratosis. Castellani and Chalmers state that they have observed the presence of a large number of polychromatic cells of different sizes, larger than normal erythrocytes, and some distinctly smaller. Some of the bodies show peculiar chromatin dots, and in microscopic specimens in which these peculiar cells are found treponemata are always present.

Like syphilis, yaws may be divided into primary, secondary and tertiary stages. There is considerable dispute as to the primary lesion. Most tropical observers of large experience are of the opinion that the first yaw is the primary lesion, and that the subsequent eruption is a secondary manifestation. Yaws is not a disease which covers any distinct period of time. Visible evidences of yaws may last from a few weeks to years, and even throughout a lifetime, if the disease remains untreated. However, there may be periods during which it would be difficult to demonstrate the lesions.

The *incubation period* of yaws is from two weeks upward although it is rare for more than five weeks to elapse after exposure to the disease. The appearance of a primary lesion is usually preceded by pains in the muscles and joints, headache, irregular temperature, anorexia, and a feeling of ill-being. In brief, the *symptoms* are similar to those in the contraction of any acute infection. The onset is followed within a period of a week by a papule. This is apparently pushed up from the rete Malpighii through the epidermis, which breaks over the summits of the papule and splits into radiating lines from the center. When the papule reaches an elevation of about a millimeter above the surface a yellow point may be observed at the apex, which consists of pus confined under the epidermis. A hair frequently projects through the center of the little pustule. This papule may result in a typical yaw, or further growth may be entirely arrested. If growth continues it results in a rounded excrescence, which may vary from a few millimeters to several centimeters in diameter. It is reddish bronze in color, and formed somewhat like a cauliflower. The name cauliflower excrescences is fre-

quently applied to a yaw. The lesion is distinctly crust-like in character. It is usually irregularly round in outline, and may rise a centimeter above the surface. The primary lesion is not indurated, but frequently painful; in the later stages, however, it is quite painless. The primary lesion may be located on any part of the body, although in males the anal region, and in females the mammary region is most common. In some countries, in which it is customary to carry children on the hip, mothers receive the infection at the point where lesions on the limbs of the child come in contact with the mother. The so-called primary lesion may heal before the general eruption begins, but, as a rule, it is present when the secondary eruption occurs. It is often stated that the primary lesion leaves a whitish scar, but this may also occur from other lesions which are present during the secondary eruption.

The secondary eruption usually begins within from one to three months after the appearance of the primary yaw, and is usually preceded by the same constitutional symptoms which occurred just prior to the appearance of the primary lesion. The secondary eruption is very similar to that of the primary lesion. There first appear minute roundish papules several millimeters in diameter. These occur on various parts of the body. There is the same formation of pus. There is considerable difference in the rate of development of the eruption, some remaining in the papular stage for a considerable period of time, and some developing rapidly into the cauliflower-like appearance heretofore described. The papules gradually become absorbed, and leave furfuraceous patches. In the dark-skinned races distinct black spots remain. In the white races reddish spots remain. The eruption is not confined to any particular portion of the body, and occurs with the same frequency upon extensor and flexor surfaces. The back and the scalp, however, is often free of eruption, and most of the lesions are found on the face, legs and arms. A decided concentration in granuloma of larger size frequently occurs in the anal region. The skin lesions usually disappear after a period of from three to twelve months, although cases have been observed in which the eruption lasted for three years, and there was no evidence of its disappearance. Successive crops of lesions may appear, and thus the skin manifestations

may last for a number of years. The yaws are seldom painful unless there is mechanical interference with motion, as, for instance, between the toes and fingers. In addition to the granuloma there may be other eruptions which consist of papular, scaly, or occasionally ulcerative manifestations. At times some of the granuloma break down and ulcers result. Yaws may have a special predilection for the palms of the hands and the soles of the feet. A peculiar papule has been described as occurring in the palms of the hands. It consists of a hard epidermic plug, which falls off spontaneously, or is easily pulled out and a deep depression remains.

There may be inflammation of the periosteum, but this is not so likely to occur until after the secondary skin manifestations have disappeared. Neuritis also occurs under similar conditions. If the disease has persisted for some time it is likely that there may be some anemia. The total number of corpuscles is sometimes reduced to 2,000,000, and the hemoglobin to 30 per cent. The leucocytes usually vary from 7000 to 12,000 per cubic millimeter. There are no characteristic changes in either the red or the white cells.

As in syphilis, tertiary manifestations may not appear for many years. Many observers deny the tertiary stage in yaws, attributing the symptoms which appear to syphilis or some other affection. With the exception of gummata the tertiary lesions clinically may resemble those produced by syphilis, but seldom in the severe form or in manifestations like general paralysis of the insane. Bone changes and sinus formation are very common. In the feet this condition sometimes resembles mycetoma, and mistakes in diagnosis are common. One or both feet may be enormously increased in size, due to enlargement of the bones, as well as an extensive infiltration. Tortuous sinuses run all through the mass, and usually discharge foul-smelling pus. The condition often begins with dactylitis, and gradually all the bones of the feet may become implicated. There is sloughing, and absorption and complete disappearance of the toes, or great distortion frequently takes place. Large ulcers over the ball of the foot are extremely common. The condition is frequently mistaken for leprosy. Dactylitis of the hands is also common, and extensive ulcerations and great deformity occur. However, there is seldom

so enormous an enlargement as in the feet. Ulceration, especially necrosis of the frontal bone, are very common. The stellate scars left by syphilis are not seen in yaws.

On *x*-ray examination great rarefaction is often seen in the bones, particularly the bones of the leg. Scars resulting from ulcers frequently contract and produce serious deformities of the hands and feet. At times these are associated with bone lesions, and great distortion may take place. Bones are sometimes bent at a right angle. The extensive ulceration in some cases of yaws which have been neglected is one of the most terrible sights imaginable, especially when associated with foul-smelling discharging ulcers.

There has been considerable discussion as to whether rhinopharyngitis mutilans, or gangosa, is a distinct entity or whether it is a manifestation of syphilis or yaws. Ley, a United States navy surgeon stationed in Guam, described rhinopharyngitis mutilans as a distinct entity which failed to respond to treatment. In later years other navy surgeons at Guam gave huge doses of potassium iodid, and reported cures in all of the cases in which it was tried. As iodid of potassium is probably efficacious in yaws and in syphilis, the therapeutic test, so far as distinguishing these two diseases, is of little value.

There are no sequelæ of yaws which do not properly belong to the third stage of the disease.

During the secondary stage the diagnosis of the disease is comparatively easy. The cauliflower-shaped excrescences are not seen in any other disease. There is, however, some likelihood that yaws may be confused with syphilis. Verruca peruana is sometimes mistaken for yaws, but it is limited to certain valleys of the Andes, and occurs at elevations of 3000 to 10,000 feet. The work of Strong and his co-workers, in which a definite organism for verruca was demonstrated, also eliminates this disease.

Some authors have regarded frambesia and syphilis as different manifestations of the same disease. Others have pointed out that syphilis occurs in all latitudes and climates, whereas yaws has been observed only in tropical countries. Castellani has reported that patients suffering from syphilis may contract yaws, and patients suffering from yaws may

contract syphilis. He also reports that monkeys successfully inoculated with yaws do not acquire any immunity against syphilis. Adequate data, however, on these points are not available.

The primary lesion in yaws is practically always extragenital. The secondary manifestation of yaws is the characteristic yaw, while in syphilis there are mucous patches, a great variety of skin lesions, loss of hair, and many other symptoms not seen in yaws. The diagnosis may be strengthened by finding the *Treponema pertenue*.

The prognosis of yaws cannot be regarded as serious, and it is extremely doubtful whether any deaths can be definitely attributed to yaws in the secondary stage. Hospital statistics have been cited among which deaths are reported, but other causes were not satisfactorily excluded. The long duration and the possible tertiary manifestations of yaws make it rank as a serious malady.

TREATMENT.

Since the advent of salvarsan and the products closely allied to it the treatment of yaws has been greatly simplified. Iodid of potassium or other drugs are no longer needed. Ehrlich's compound, with its variations, is much nearer a true specific for yaws than it is for syphilis. Usually an intravenous injection of 0.6 gram (10 grs.) of salvarsan is sufficient to bring about a rapid cure. However, it has been deemed advisable to repeat the treatment at least once or twice, in order to guard against the possibility of tertiary manifestations. The rapidity with which salvarsan causes the skin eruptions in yaws to disappear is almost unbelievable. Extensive granulomatous masses all over the body are frequently absorbed, and the skin, in so far as discolorization is concerned, may return to normal within a period of two weeks. The technic for injection should be the same as for syphilis. As intravenous injections are the most effective, other modes of administration should be disregarded. The various local antiseptic washes which have been recommended are now seldom necessary. In the tertiary stage, amputation of the hands or feet is sometimes indicated. An artificial limb is often more serviceable than the deformed natural member.

As a prophylactic measure, persons residing in areas in which yaws occurs should be careful to prevent solution of continuity in the skin, and when abrasions do occur a protective collodium or proper dressing should be immediately applied. Doctors, nurses, and others coming in actual contact with yaws cases should immediately disinfect their hands thoroughly after having been exposed. Persons suffering with yaws should be isolated in separate rooms, and have their own toilet articles and food utensils.

ORIENTAL SORE.

Oriental sore is a specific ulcerating granuloma of the skin, caused by *Leishmania tropica,* and the disease occurs in circumscribed areas in tropical countries. This type of tropical ulcer is also known as Delhi boil, Bagdad boil, bouton d'Orient, chancre du Sahara, endemische Beulenkrankheit, Aleppo boil, Biskra boil, yearly boil, granuloma endemicum (Brooke), Sudan nodules, Leishmaniasis (Wright), and Leishman nodules.

Descriptions of the disease are available as early as the eighteenth century. With the advent of bacteriology, many organisms have been described by different authors as the cause of the disease. However, it was not until 1903, when Wright described bodies similar to those found in kala-azar, that the etiology of Oriental sore was placed upon a definite basis. Wright's work has been frequently confirmed by workers in tropical diseases.

Oriental sore occurs in many tropical and subtropical countries. It is especially prevalent, however, in Morocco, Tunis, Tripoli, Egypt, Crete, Congo, Syria, Asia Minor, Mesopotamia, Persia, and many parts of India. Occasionally cases are reported from Brazil, British Guiana, and in temperate climates. A case was described as having occurred in Boston a few years ago.

The incubation period may be days, weeks, or months. The short incubation period is established by the appearance of the disease in a few days in new arrivals in endemic areas.

Oriental sore is caused by the *Leishmania tropica* (Wright). In 1908 Nicoll first cultivated the parasite on blood-agar. It

grew slowly, and produced flagellate and division forms on the fourth day, and by the tenth day rosettes appeared which were visible to the naked eye. A typical flagellate measures from 40 to 45 microns, with a breadth of 2 to 4 microns. The organism is apparently exactly the same as that of kala-azar, although it gives rise to no constitutional symptoms which resemble that disease. The Oriental sore has been successfully inoculated into monkeys from cultures.

In the common ulcerative variety there is usually atrophy and disappearance of the epithelium, with extensive cellular infiltration of the corium and papillæ. Many interpapillary down-growths occur in the rete. There are usually vertical epithelial columns extending deeply downward, with a few cells and isolated masses of cell infiltration. The sore may vary from 5 or 6 to several centimeters in diameter.

The disease may be transmitted from person to person by absorption of the virus through an abraded surface, or through small wounds or ulcers. Insects, especially flies, have been suspected of transmitting the disease. It is inoculable from man to man. Whether there is a definite life cycle of the organism in the fly or mosquito has not been satisfactorily demonstrated, although it is more than likely that these insects are at times concerned in the transmission of the disease.

The sore begins with a minute itching papule, which has a tendency to increase in size. In a short time, which period may vary from a few days to several weeks, the papule becomes covered with fine paper-like scales. A crust soon results, and if this is removed a sore is found underneath. This soon increases in size, and discharges a scanty, ichorous material. This discharge at times may undergo crust formation, and the discharge temporarily ceases. Extension of the ulcer takes place by erosion inward, and there are sharp-cut jagged edges. Granulations speedily break down. Subsidiary ulcers may occur around the parent ulcer, with which they all ultimately merge. In untreated cases healing may begin in a few months, but it may extend over a year. Ultimately a cicatrix forms, which, upon contraction, produces various deformities. The ulcers of the face are often severe, and may be single or multiple. No very characteristic blood changes

have been reported. The ulcers usually occur upon the uncovered portions of the body, and are very frequently upon the hands and feet. They are seldom seen on the palmar surfaces of the extremities or in the scalp. There seems to be no special race immunity, all classes being prone to be attacked. Various types of the disease have been described. The common variety consists of several nodules which ulcerate slowly, and in some cases there is fever and enlargement of the spleen. There is a verrucose type, which has been described by Ferguson and Richards. There is also a non-ulcerative variety, characterized by the presence of pinkish nodules which never seem to ulcerate.

When the disease occurs in endemic form the diagnosis is not very difficult. This may depend upon a few eruptive elements, often only one lesion, situated on an uncovered part, or in a *characteristic papule which slowly enlarges* and gradually undergoes ulcer formation. The presence of the *Leishmania tropica* micro-organism is characteristic and sufficiently diagnostic in itself.

The prognosis is excellent so far as life is concerned. At times the sores may become phagedenic.

TREATMENT.

Treatment, on the whole, has been rather unsatisfactory. The destruction of the ulcer with a caustic has been recommended. It is doubtful whether these are of any great value. Antiseptic applications and frequent cleansings are indicated. Cases observed in the Tonga Islands healed quickly by thorough irrigation with bichlorid of mercury solutions and dressings of wet bichlorid. Ulcers that have been actively treated for a few weeks should be treated with a benign ointment like vaselin or lanolin, to be followed later with the stronger antiseptics. Tonics for those who are anemic or otherwise below par are rational therapeutic accessories.

SPRUE.

As synonyms for this infection the terms tropical diarrhea, tropical aphthæ, Ceylon sore mouth, Cochin China diarrhea, and spruw (Dutch) are generally employed.

Sprue is a disease which manifests itself by chronic patchy inflammation of the gastrointestinal tract, resembling aphthous stomatitis. It is characterized by the passage of periodic, copious, whitish, frothy, fecal discharges.

The disease was described in 1766 by Hillary, of Barbados, under the name of aphthoides chronica. Later descriptions were contributed by observers in India and Java. More recently the disease has been described by the French in Cochin China. In 1880 Manson clearly defined the disease, and somewhat later an excellent description was given by Van der Burg. Since the descriptions of Manson and Van der Burg, sprue has been recognized as a definite clinical entity, and descriptive articles have been numerous. More recently Ashford, of Porto Rico, has made extensive contributions, and several British writers also have studied the disease at length.

Sprue is found in tropical and semitropical countries, but no cases have been found in permanent residents of the temperate zone. The disease occurs in the West Indies, Malaya, Sumatra, Java, Siam, Cochin China, Ceylon, India, the Fiji Islands, the Philippines and Korea.

Residence for several years in countries in which sprue occurs seems to be one of the essentials to contracting the disease. Exhausting gastrointestinal affections—for instance, the diarrheas and the dysenteries—may terminate in sprue. Other conditions which lower the vitality also are considered as being important predisposing elements. Specific micro-organisms have been described from time to time, but none of them has as yet received universal acceptance. One of the latest of these is a monilia described by Ashford,[103] of Porto Rico. He reports that in pure culture 5 drops injected into the muscles of a Belgian hare caused death in seventy-five hours, with enormous production of intestinal gas and diarrhea. Also that the germ was recovered from the spleen, liver, and other organs, in pure culture a few hours after death. He also reports that this monilia can be regularly found in bread.

Pyorrhea alveolaris seems to be associated with nearly every case. This condition, however, is very common in the tropics among people who do not have sprue.

The deficiency theory of the cause of sprue has received considerable attention, and has been ably presented by Cantlie.

The writer has seen no cases of sprue in countries like the Philippines, India, Ceylon, the Fiji Islands, and Sumatra, among natives, although it was quite common among Europeans.

With the exception of the lesions found in the intestinal tract, there are no important changes in the remaining organs. As a rule, the tissues are abnormally dry. Fat is completely absent. The muscles and viscera are, as a rule, anemic and atrophied. Occasionally there is fatty or granular degeneration of the pancreatic cells.

The intestines are extremely thin and almost translucent. The serous coat is not much changed. The muscular coats are atrophied. In some areas the mucosa shows fibrous change. The mucous membrane, from the mouth to the anus, usually in patchy form, is superficially eroded and interstitially atrophied. The interior of the intestines is coated with a thick layer of grayish, sticky mucus overlying patches of congestion, erosion or ulceration. Frequently there are also found pigmented areas and thin scars or cicatricial patches. Villi are often completely destroyed. Small nodular indurations several millimeters in size are frequently seen. These, on section, are found to be minute, cyst-like dilatations of the follicles, and contain a mucopurulent material. As a rule, the erosion is most marked in the lower part of the ileum and the upper part of the large bowel. The mesenteric glands ordinarily are large and pigmented.

In the absence of definite etiology the pathology must be largely speculative. Whether the first pathologic changes are due to physical exhaustion, whether the disease depends upon a specific organism or upon deficiency causes, or whether there is a combination of these factors, is unknown.

Hyperactivity of the liver may be regarded as one of the first steps in the development of the disease, and this soon results in exhaustion of the hepatic function. Similar hyperactivity in other digestive glands is also usually assumed. This results from chemical changes in the food, with the resultant formation of chemical changes leading to the conditions which produce the fermentative diarrheas. Analysis of stools indicates the presence of the ordinary elements of the bile, notwithstanding the fact that pale stools would indicate

their absence. *Post-mortem* examinations show that the feces are usually normally bile-stained in their upper portion, and gradually become pale as the rectum is reached. Apparently bile is formed, but no bilirubin. There is excess fat in the stools, the causation of which is not clear. Halberkaun[104] states that instead of the normal 6 to 8 per cent., the fat varies from 20 to 40 per cent., and that this is not due to failure of the fat-splitting enzymes, but to faulty absorption in the upper intestine.

Anemia, especially in the later stages, is one of the characteristics of the disease. There is great reduction in the number of erythrocytes, and the hemoglobin index is low. There is no great change in the number of leucocytes. There may be an increase in the proportion of lymphocytes.

Attacks of sprue vary greatly in intensity. There may be only slight periodic digestive disturbances, with small erosions in the mouth or evidences of pyorrhea alveolaris. On the other hand, there may be extensive erosion of the mouth and of the intestinal tract, with acute pain when food is taken. The symptoms may vary between these two extremes, and any combination is possible. Sprue may continue for one or two years, or it may be extended for ten years or longer. Death finally comes from complete exhaustion. The disease is undoubtedly greatly modified by treatment and climate. In a typical case the patient, as a rule, soon becomes extremely emaciated. The complexion is dark, sallow and yellowish. The principal symptoms complained of are sore mouth and digestive disturbances, with distention of the abdomen and looseness of the bowels, especially soon after rising. Soreness extending from the mouth to the anus is very characteristic of the disease. The patient is physically weak, often has loss of memory, and is unable to take physical exercise, or to be capable of continuous application. Irritability and unreasonableness are common. On examination of the mouth the soreness complained of will be found to depend on numerous lesions of the mucous membrane. The patchy erosion appears superficial in character. There is considerable difference in the severity of the mouth symptoms. At times they may completely disappear for several weeks, and may be followed by an acute exacerbation. The tongue is extremely red and raw-

looking. There is extreme tenderness of the gums. The tone of the tongue is excellent, and it is usually markedly pointed on protrusion. At times there may be minute vesicles on its surface. Often the erosion extends to the lips, and vesicle formation is common. The mucous surfaces of the cheeks also show erosion. Bleeding does not occur very often. An attack of sprue usually begins with sore mouth, indigestion and morning diarrhea. These frequently disappear, and the patient does not recall having had them until direct attention is drawn to their occurrence. Gradually the attacks increase in frequency and in severity. If the teeth are roughened, especially the molars, ulcers may form at the points of friction with the cheeks. The mouth is often very tender, and deglutition and mastication become difficult. Warm and spiced foods or alcohol may cause considerable pain. On swallowing, a burning pain is frequently felt along the line of the esophagus. There is usually a sensation of discomfort and distention after meals, with eructations. Vomiting is rather frequent. After the bowels have moved once or twice the patient usually feels considerably relieved during the remainder of the day. Borborygmus is very common. The symptoms usually become very much more pronounced after ingestion of a full mixed diet. As a rule, great relief is obtained by a close adherence to a milk *régime*.

The diarrhea may vary considerably in type. There may be a regular morning diarrhea, which persists for weeks, followed by a rest period of several weeks. The quantity of the stool is at times enormous, and this symptom is one of the most characteristic signs of the disease. The stool consists of pale, grayish, pasty, fermenting, evil-smelling masses, accompanied by copious amounts of frothy and watery material. The excreta usually contain masses of undigested food and large amounts of oil and fatty acids. On voiding the stool there is considerable relief from the distention. The stools are usually most active during periods in which the erosion of the mouth is the greatest. Microscopic and chemic examination of the stool shows mucus, epithelial *débris*, many bacteria, frequently yeast-like fungi, and often the eggs of intestinal parasites. Analyses carried on by Harley and Goodbody showed that when 12.99 grams of nitrogen were adminis-

tered, 1.47 grams of nitrogen were recovered from the stools, thus showing that 88.86 per cent. had been absorbed. When 76.44 grams of fat were administered, 35.92 grams were recovered, showing that 53.01 per cent. had been absorbed.

The blood shows a low color index, with the erythrocytes varying, as a rule, from 1,000,000 to 3,000,000 per cubic millimeter. There may be a slight reduction in the number of leucocytes. There are no characteristic changes in the urine. As the disease progresses the patient becomes emaciated, weak physically, and depressed and irritable mentally. The skin shows lack of proper nutrition, and is dry and rough. The patient continues to lose weight, and typical cases of sprue are mere skeletons. The patients are seldom ill enough, however, to take to their beds, and most of them drag out a miserable existence. It is essentially a chronic disease. Death usually comes from exhaustion. During an acute attack cardiac failure often results. As a rule, there is no elevation of temperature. The pulse is that found in ordinary weakened conditions, and is not especially characteristic of sprue. Attacks of dysentery are often followed by sprue. In this condition the stool gradually changes from the typical dysenteric stool to that described for sprue. It has been observed lately that pyorrhea alveolaris is usually associated with cases of sprue, but it is not known whether it is directly associated with the disease. At times there are cases of sprue without diarrhea, the diagnosis being based upon the sore mouth, the distention of the abdomen, the anemia, and the copious stools, with their sprue characteristics.

In some cases which have recovered, permanent digestive disturbances remain due to glandular lesions resulting in atrophy. Patients of this class may be condemned to a life of dietary restrictions.

Severe hemorrhages sometimes accompany the attacks. Insomnia is frequent. Infections with intestinal parasites, especially the hookworm, are also common. Chronic appendicitis may also result. Jaundice occurs in a small percentage of cases.

In well-marked cases of sprue the *diagnosis* presents no great difficulty. The sore mouth, the intestinal distention, the

diarrhea, and the characteristic large, fermentative stools are sufficient data upon which to base a correct diagnosis. The raw sore condition, which extends from the mouth to the anus, is very characteristic, and is seldom seen in any other disease. There is no other diarrheal disease which has stools resembling those found in sprue. The characteristic mouth lesions, with the periodic remissions, are also very distinctive. The large percentage of fat found in the stools is also of considerable diagnostic importance. Ordinarily stomatitis may be excluded by the absence of the characteristic discharges. A similar distinction applies to thrush. Hill diarrhea is sometimes confused with sprue, but in this disease there are none of the characteristic mouth lesions. Sprue, however, sometimes follows hill diarrhea. Dysentery may be readily distinguished from sprue by the presence of the causative organisms of the various forms of dysentery.

The prognosis in sprue, with a change of climate to temperate regions and a strict adherence to diet, is reasonably fair, and fatal endings rarely occur. In cases which have existed for six months or more without treatment, or change to a favorable climate, the prognosis is not so good.

TREATMENT.

The many treatments which have been advocated for sprue are legion, and vary enormously in extent. None of them can be said to be very satisfactory. However, strict adherence to the milk diet is one of the treatments used in the past which has given the most consistently favorable results. The thorough co-operation of the patient is essential. Everything which can be done to conserve the energy of the patient is of direct assistance in bringing about a recovery. A change to a temperate climate is of great importance, although many cases recover in the same environment in which they contracted the disease. The various diets which have been prescribed may readily be divided into the following classes:

1. Milk diet.
2. Milk and fruit diet.
3. Fruit diet.
4. Meat diet.
5. Meat and milk diet.

A careful survey of the cases and treatment of sprue during the past few years in the Philippines, India, Sumatra, Fiji Islands and Ceylon, and of those being treated in the United States shows rather conclusively that the milk diet gives the most consistent relief, and results in the greatest percentage of recoveries. Recently Lunn,[105] of Manila, has reported success by the use of emetin, neosalvarsan and sodium cacodylate. Lunn administers 0.9 grams (15 grs.) of neosalvarsan intravenously. The diarrhea, he reports, usually ceases within two days. The patient may remain apparently well for about a month, when the symptoms return. Neosalvarsan is again injected, and recurrences do not take place. He reports one patient as having gained twenty-five pounds a few weeks after this treatment was used. In cases in which there is considerable pyorrhea alveolaris a combination of sodium cacodylate and emetin hydrochlorid seems to produce favorable results. A dose of 0.05 gram (⅞ gr.) of sodium cacodylate was administered in a 5-mil (1.35 f3) solution into the gluteal muscle at daily intervals for three days. The daily dose was then increased to 0.10 gram (1.54 gr.) for an additional ten days. One-grain (0.06 Gm.) doses of emetin hydrochlorid were given daily until twenty doses had been taken. In some instances he has found that alternate daily doses of the sodium cacodylate with the emetin hydrochlorid give the best results. The treatment, however, should not be prolonged beyond twenty doses for each drug. It may be possible that the emetin produces favorable results owing to the accompanying pyorrhea alveolaris. The teeth should be put into good condition by a competent dentist. Wright has recommended succinimid of mercury in pyorrhea alveolaris, and reports a small number of cases in which it proved of value. It may be possible that this drug might also prove valuable in the treatment of sprue, and a thorough trial seems to be indicated.

Silver nitrate may be used in treating the ulcers in the mouth, and this often seems to have a beneficial effect on the disease.

Wherever possible, rest in bed with special attention to building up the physical condition should be insisted upon. In adopting the milk diet, modifications to suit the patient

must be worked out for each individual case. Safe fresh cows' milk is desirable, but boiling or pasteurizing does not seem to be any detriment, and if there is any suspicion as to the wholesomeness of the milk this precaution should always be carried out. The milk should be administered, preferably, warm, and in small quantities. More or less continuous sipping is the most effective mode of administration, and the patient should be encouraged to take daily amounts up to seven or eight pints. Other modifications may be necessary, as, for instance, additions of Vichy water or small quantities of salt, or other formula may have to be adopted to suit the individual needs of the patient. If all forms of alimentation except milk can be eliminated it will, in all probability, result in the earlier cure of the case. Brown[106] reports that he controlled the diarrhea with 30-grain (2 Gms.) daily doses of pancreatin, combined with calcium carbonate and tannic acid.

With regard to prophylaxis nothing definite can be said under this head. It would seem desirable, however, to provide the patient with separate table utensils, which are thoroughly disinfected after every meal. The stools, as soon as voided, should be incinerated. If that is not possible, they should be disinfected with a 5 per cent. solution of carbolic acid, or 1:500 bichlorid of mercury, or 1:500 solution of phenoco. After touching the patient or his discharges the hands should be disinfected.

LEISHMANIASIS.

Kala-azar is a communicable disease, chronic in type, with irregular fever and enlargement of the spleen, in which there is found the Leishman-Donovan body. It is also known as kala-azar, dumdum fever, cachectic fever and kala dukh.

One of the first records of the disease appears in the Assam Sanitary Report of 1882, in which attention is drawn to the fact that the administration officers in Assam as early as 1869 reported a peculiar disease referred to by the natives as kala-azar. In some sections of Assam entire districts were depopulated by its ravages. It was soon observed that the disease spread along the lines of human travel. For many years it was regarded as one of the severe forms of malaria,

but the malarial parasite could not be demonstrated. Giles, in 1889, after an extended study of the disease, concluded that it was due to the ancylostome, because he found the ova of the hookworm in the feces in practically all cases which he investigated. The theory, however, did not receive universal acceptance. Dobson created considerable doubt as to the correctness of the theory when he showed that he had found 75 per cent. ancylostome infection in 212 cases of other illnesses, and that in 146 apparently healthy men he had found an ancylostome infection of 67.12 per cent. Rogers and Ross reinvestigated the disease between 1896 and 1898, and came to the conclusion that it was malarial in nature. Rogers regarded it as the malignant type of malaria, and Ross as an

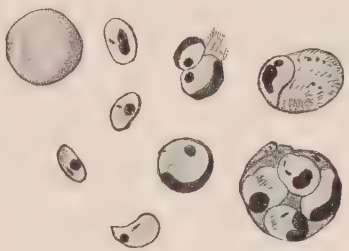


Fig. 14.—Leishman-Donovan bodies. (*Da Costa.*)

infection secondary to malaria. Bentley, in 1903, reported it as a malignant form of Malta fever. In 1903 Manson suggested that the disease was probably due to trypanosome infection. Shortly afterward, in the same year, Leishman published a report in which he stated that at a *post-mortem* of a soldier who died in 1900 of dumdum fever he had discovered in smear preparations from the spleen a number of small round or oval bodies, which, upon being stained, showed a nucleus and a small, rod-like, chromatin mass set perpendicularly to the circumference of the larger nuclear mass. In the same year he found similar bodies in a rat which had died of nagana, the blood of which, during life, had contained trypanosomes. He then surmised that the bodies found in the soldier probably represented the degeneration forms of trypanosomes. In the same year Donovan stated that, three months previous to the date of publication of Leishman's

report, he had found similar bodies in smears from the spleen taken *post-mortem* from cases said to have died from chronic malaria. Later he found identical bodies in blood taken from the spleen during life from a patient suffering from irregular fever and enlarged spleen, in which no malarial parasites could be found. Earlier in the year Marchand found similar bodies in sections of the spleen, liver and bone-marrow, in a patient who had been in the Peking campaign. He had been ill for a long time with continued irregular fever accompanied with great enlargement of the spleen and anemia. After these reports were published many investigators in different parts of the world reported the presence of these bodies. Wright, of Boston, found parasites morphologically indistinguishable from those found in Leishmaniasis in the granulation cells of Oriental sore. This latter observation has been frequently confirmed.

The disease has been more frequently reported from India than from any other country, especially from Assam. The Garo hills is the district in which it first attracted attention. It has also been found in at least four of the provinces of China, in Arabia, Ceylon, Indo-China, the Sudan, Algeria, Crete, Sicily and South America.

The consensus of opinion is that the disease is caused by a *Leishmania donovani* parasite, and that it is transmitted to the human body by some insect. There is considerable difference of opinion as to whether the parasite belongs to the herpetomonas group or to the trypanosoma group of protozoa. Many observers believe that the parasite is transmitted by the clinocoridæ. Price and Rogers[107] report that uniform success in prophylactic measures may be had which is based on the observation that the infection persists in houses and their sites. Infection can practically always be stopped by removal to new quarters three or four hundred yards from infected houses, and they conclude that if the disease were transmitted by flying insects, as mosquitoes or flies, removal to this short distance would not be effective. In their experience a change of habitations had no effect in controlling malarial fever. From the foregoing they conclude that the disease is transmitted by a non-flying insect carrier, which is probably the bedbug. Measures for the eradication of this insect prevent

the appearance of new cases. In India the disease spreads very slowly along the lanes of human travel, and often appears after the introduction of a new individual who has lately lived in endemic areas. Apparently the disease persists in a given community for about six years, and then disappears, even though no control measures have been invoked.

According to Rogers the parasite of Leishmaniasis belongs to the genus *Herpetomonas leger*. There are two stages, the intracorporeal and extracorporeal. It has been suggested that one of these represents the sexual and the other the asexual form. The latter is found in man, and, possibly, some other vertebrates, while the former occurs in certain insects, as flies and bugs. The parasite is distributed throughout the body, but it may be found most frequently in the endothelial cells of blood-vessels and lymphatics, and can nearly always be obtained by puncture of the spleen. It also occurs in the blood, being found in the polymorphonuclear and the mononuclear leucocytes, and at rare intervals in the erythrocytes. It occurs most abundantly in the blood toward the termination of the case. The organism is a small, ovoid body, measuring from 2 to 4 microns in diameter. When stained in accordance with the Leishman method it shows lavender-colored chromatin masses, one larger than the other, inclosed in the cytoplasm, which stains a bluish tint about the periphery. The smaller chromatin mass is the micronucleus, and is usually the shape of a short rod and is placed perpendicularly or at a tangent to the nucleus; and, also, stains more deeply than the nucleus. Multiplication in the body takes place by simple fission. The nucleus and centrosomes divide first. The parasites are intracellular, and the cell is gradually distended by the multiplication of the bodies until it finally bursts, after which the bodies attack other cells or are engulfed by the white blood-corpuscles. In smear preparations, however, they may be found free or in clusters. Sometimes several hundred parasites may be found in a single field. In culture media the parasites enlarge rapidly. The cytoplasm becomes granular, opaque and vacuolated. The parasites may enlarge up to 9 microns, after which they assume elongated, piriform shapes, and become flagellated. The flagellum occurs at the rounded end of the parasite, and projects from the body.

This flagellum measures from 15 to 20 microns in length, and multiplies by longitudinal fission. All attempts to transmit the parasite to vertebrates have failed.

Leishmaniasis attacks both sexes and all ages, but shows a decided predilection for those who are acclimatized. In other words, it is more severe among old residents than among new arrivals.

After the parasite is introduced into the body, multiplication takes place rapidly in the endothelial cells of the blood-vessels and lymphatics. The organs most affected are the liver, spleen, bone-marrow and the lymphatic glands. The parasites then rupture the membrane in which they are inclosed, and are engulfed by the leucocytes. Another stage in the development probably takes place in an insect host. There is a marked change in the leucocytes; also a very definite anemia. The erythrocytes may be reduced to 2,500,000, with a proportionate reduction in the amount of hemoglobin. Leucopenia is definite, and in some cases the number of leucocytes may be reduced to 1000. There is great diminution in the percentage of the polymorphonuclear leucocytes. This is supposed to predispose to bacterial infections.

As a rule, the body is very much emaciated, with marked muscular atrophy. There is enlargement of the spleen, and often the liver may be swollen. Sometimes there is ulceration of the skin and intestines. The spleen is firm, deep red in color, and often shows malarial pigment. The capsule is thickened, and the trabeculæ are increased in size. On section in smear preparations large numbers of parasites may be found. The intralobular capillaries of the liver are dilated and contain macrophages. The bone-marrow contains numerous parasites, and the yellow marrow is usually red. Parasites can frequently be found in the ulcers and papules in the skin.

The *incubation period* is unknown. Cases have apparently occurred within ten days after exposure. The disease is usually ushered in with a decided chill, which recurs at daily intervals. This is accompanied by an irregular, high, remittent fever, with two remissions during a twenty-four-hour period. Rogers regards the double remission as one of the most important diagnostic signs. Between the third and sixth

week the temperature declines, gradually reaches normal, and the first febrile attack of the disease terminates. The spleen and liver become enlarged, and are painful and tender. A headache is frequently present, but it is not severe. This condition is followed by a period of complete apyrexia and general improvement. Recurrences may take place at intervals of a month or more. No drugs appear to have much influence on the attacks. These recurrences become more and more frequent, until fever is present more or less all the time. During the remissions in temperature there is profuse sweating. Pains in the limbs are referred to as similar to those in rheumatism. The facies in kala-azar are very characteristic. There is an anxious, apprehensive look. After the disease has persisted for some time there may be edema of the legs, cystitis or circumscribed edema. The skin in white persons often acquires a muddy-gray color. The hair loses luster and becomes brittle. Petechial spots are common, as well as epistaxis and bleeding from the gums. Fever and enlargement of the spleen and liver may continue for months, or even for periods as long as two years. Intercurrent diseases are common, and many patients die of dysentery, tuberculosis or pneumonia. Marked changes in the blood may be observed. There is a decided decrease in the number of both red and white cells, with a constant reduction in the number of leucocytes. The tongue is usually clean, and the appetite remains good. The abdomen is swollen and barrel-shaped. Gentle palpation reveals the spleen well below the costal margin, and often as far down as the pubis. It is usually possible to outline it by a percussion. The liver is also frequently enlarged. When the liver and spleen are enlarged there are often intestinal disturbances which manifest themselves as diarrhea or dyspepsia. Papular eruptions are frequent on the thighs. There is often edema of the lower extremities, especially the ankles.

As complications, intercurrent infections are common, and pneumonia, tuberculosis, diarrhea, septicemia, and other bacterial invasions are frequent.

A remittent fever of several weeks' duration, followed by apyretic periods and finally by continuous chronic temperature, and enlargement of the spleen and the characteristic

facies are sufficient, in most cases, to make a reasonably accurate diagnosis. This, however, can be confirmed by demonstrating the Leishman-Donovan bodies. If the bodies cannot be found in the blood, punctures from the spleen frequently reveal them. Before the puncture of the spleen is undertaken most rigid aseptic precautions should be observed. Many unfortunate accidents have occurred in attempting the spleen puncture. At times leukemia may be confused with Leishmaniasis, but the characteristic blood changes in leukemia are sufficient to distinguish between the two diseases. Malarial fever also at times confuses the diagnosis, but the characteristic blood-parasites serve to distinguish the two. Typhoid fever may be distinguished by the presence of Widal reaction, and the characteristic step-like rise in temperature without the marked remissions which occur in Leishmaniasis. Advanced cases of ankylostomiasis may also be confused with Leishmaniasis. Appropriate treatment for those who have ankylostomes in the stools usually brings about prompt relief, and the fever seldom is prolonged as in ankylostomiasis.

Until recently the prognosis of kala-azar has been grave. From 70 to 95 per cent. of all cases died within a period of two years. The treatment recently instituted, however, bids fairs to reduce this high mortality.

TREATMENT.

Rogers[108] has recently reported considerable success with intravenous injections of tartar emetic. He recommends the use of ½ mil (8 *m.*) doses of 2 per cent. solution of tartar emetic every two or three days up to tolerance, which is from 3 to 4 mils (48.6 to 64.8 *m.*). If gastric disturbances occur, the dose is slightly reduced. No serious symptoms occurred when the larger dose was given. The treatment is continued until the temperature has remained normal for several weeks, with continuous gain in weight and the disappearance of the parasites from the spleen. It is not necessary to continue treatment until the spleen becomes normal in size. In successful cases the spleen diminishes in size after treatment has been stopped. Quinin is apparently useless, unless there is an accompanying malaria. Rest in bed, liquid diet, and the treatment generally applied to typhoid are applicable during

the fever periods. Manson has reported 2 cases out of 4 as being benefited by intramuscular injections of atoxyl. He administered it in 3-grain (0.19 Gm.) doses for three days for a period of a year or longer. Attention should also be directed toward relieving the patient of intestinal parasites and building up the general health by change in climate, good food, and rest in bed.

Rogers has made extensive investigations into the prevention and control of Leishmaniasis, and reports satisfactory results by vacating buildings in which the disease has occurred. The disease disappeared repeatedly among laborers who were transferred from infected buildings to uninfected quarters. He attributes the success of the measures to the elimination of bedbugs and similar insects.

TYPHUS FEVER.

Typhus fever is an acute infectious disease of doubtful origin, transmitted by pediculi, characterized by sudden onset, discrete maculated rash, and fever terminating by crisis usually at the end of the second week. Spotted fever, ship fever, jail fever, exanthematic typhus, tabardillo (Mexico), and camp fever are the ordinary synonyms for this disease.

It is probably one of those diseases that has been frequently confused with plague during early historic times. During the nineteenth century it was constantly confused with typhoid fever. The word signifies smoke or mist in Greek, and was used by Hippocrates to describe any febrile condition in which stupor was a prominent symptom. The term typhus was first applied to the disease by Boissier de Sauvages in the eighteenth century. It came into general use through its adoption by Cullen. Typhus has also frequently been mistaken for relapsing fever. The disease is now known to be endemic in Northwest India, Mexico, Russia, the Balkans, Japan, China, and, no doubt, in many other places where poor sanitation prevails, and where body lice are common. Epidemics occurred in New York in 1881 and 1882, again in 1892 and 1893, and in Philadelphia in 1883. Brill[109] first described a disease resembling typhus as occurring in New York in 1896, and since then he has made frequent contribu-

tions on its presence. It has since been shown by Anderson and Goldberger that Brill's disease and typhus have the same etiology; the former, however, is much milder in character. The disease has caused terrible havoc on immigrant ships, in jails, or other places where human beings are compelled to share quarters under crowded conditions.

Typhus fever is primarily a disease of temperate and cold climates, but there have been frequent outbreaks in high altitudes in the tropics. It is a disease of winter, and disappears with remarkable regularity upon the approach of warm weather. For instance, in Mexico during the past few years, probably epidemics of 100,000 cases or more have occurred; but these promptly subside on the approach of warm spring days. In Mexico the disease prevails almost entirely in the high plateau, most of which is from 4000 to 7000 feet above sea level. An outbreak occurred in the Philippines in 1913 in the highlands of the Island of Mindanao (altitude, about 3000 feet). It was probably conveyed there by Japanese laborers recently arrived from Japan. Typhus had been prevailing in epidemic form in Japan just previous to its appearance in the Philippines. Severe epidemics have occurred since the outbreak of the European war of 1914. Serbia is probably the country which has suffered most. The outbreak in Serbia was so severe that physicians from most of the civilized countries of the world answered the call to go to Serbia to assist in bringing the disease under control; many physicians and nurses succumbed to the disease. The relief measures were probably not well started until the approach of spring, which makes it likely that the disappearance of the disease was due more to warmer weather than to sanitary measures. It is, nevertheless, true that the measures which were applied to destroy vermin in hospitals and other infested human habitations must have had considerable influence in reducing its ravages. The disease has also prevailed extensively among the Austrian and Russian troops. Typhus has been repeatedly carried into Germany, and as a defense measure stations with steam chambers have been established between Russia and Germany, and all persons desiring to enter Germany must pass through one of these *entlausung* stations, and a certificate obtained to show that they are free from vermin.

At the present time there is much dispute with regard to the etiology of typhus fever. Plotz,[110] of Mt. Sinai Hospital, in New York, has isolated an anaërobic organism which he claims is the true etiologic factor. He has made serums for the cure of the disease, but the reports so far published are not especially encouraging as to their value. Other observers state that organisms similar to those found by Plotz can be recovered with the same technic in other febrile conditions.

The discovery of the typhus organism has heretofore been frequently announced. Klebs[111] found bacilli in 1881. In the same year Mott and Blore[112] described a minute, screw-like, motile organism, as being present in the blood during life, and certain micrococcus bodies in the muscular fibers of the heart after death. In 1891 Hlava[113] described ovoid bodies. In 1892 Thiomot and Calmette[114] saw flagellated bodies. In brief, some research worker has announced a typhus organism almost every few years.

Nicolle, Anderson and Goldberger[115] have experimentally inoculated the disease into monkeys. In 1909 Nicolle[116] reported the transmission of typhus fever by the bite of the body louse (*Pediculus vestimenti*). This work has been confirmed by Ricketts and Wilder,[117] and by Anderson and Goldberger.[118] Anderson, Goldberger and Foster have also shown that the head louse (*Pediculus capitis*) also may be concerned in transmitting the infection. The very extensive experience in the great war warrants the deduction that the disease is conveyed entirely by vermin. Body lice are probably responsible for the great majority of cases, but other vermin, especially the head louse, may at times be responsible. The disease may occur in sporadic form, especially the mild type observed by Brill in New York. Under these circumstances recognition is rather difficult. A mild type has been reported in Manchuria. These mild outbreaks have a very low mortality. In Mexico the disease is usually virulent, and the mortality often 25 per cent. Ricketts, of the University of Chicago, fell a victim to typhus while carrying on research work in Mexico City. The disease is associated with overcrowding, and can usually be traced to conditions in which there is opportunity for persons to exchange body lice.

During epidemics it is a highly contagious disease, and non-immunes in attendance upon patients are usually attacked. Nurses, doctors, and other attendants are frequent victims. In China, among a very small group of medical men, no less than five have lost their lives during the past two years. It is stated that in Ireland, among 1230 physicians attached to institutions, 550 died of the disease.[119]

The infective agent may be found in the peripheral blood between the second and fifth days of the fever. The disappearance of the virus of the disease on the fifth day corresponds with the appearance of the eruption, and it has been held by some authors that the virus leaves the blood and enters the skin. Plotz, of Mount Sinai Hospital, New York, finds an anaërobic Gram-staining bacillus in the circulating blood, which he claims produces the disease on being inoculated in pure culture into animals.[120]

At autopsy, as a rule, no characteristic lesions are found. The organs present the appearance of an acute infection. There may be some cloudy swellings of the liver and kidneys, and moderate enlargement of the spleen. It is said that if death does not occur until after the second week, there is no enlargement of the spleen.

The *incubation period* of typhus fever is variously given from four to sixteen days. Twelve days is probably a fair average. In a large number of cases studied in Mexico last year the incubation period was apparently ten days. The invasion, as a rule, is abrupt and marked by chills, or sometimes by a single rigor that is immediately followed by fever, headache, pain in the back and in the legs. There is considerable prostration, and the patient usually takes to his bed immediately. There is a step-like rise in the temperature until the tenth to the thirteenth day, and then a fall by crisis. The low point is usually reached in twenty-four hours. A pseudocrisis sometimes occurs on the eighth or ninth day. The average high point in the temperature is between 104° and 105° F. (40° and 40.5° C.), but it may go as high as 107° F. (41.6° C.). In the mild form the temperature is much lower. In fatal cases the temperature may reach 108° or 109° F. (42.2° or 42.7° C.). The tongue is dry, swollen and cracked, and crustated with a thick, brown deposit. The tips and sides

of the tongue are red. There may be nausea. Vomiting is rare. The bowels are usually constipated. The nervous system is affected early in the disease. The patient is usually apathetic and drowsy, and has a dull expression. The delirium may vary from the mild type to that of the most severe maniacal form. The eruption appears between the third and fifth days, first upon the abdomen and upper part of the chest, and then upon the extremities and face. The eruption is most difficult to distinguish in the colored races. It usually occurs in three stages: first, erythema; second, a macular eruption which at the beginning resembles the spots of typhoid, varying from 1 to 10 millimeters ($\frac{1}{25}$ to $\frac{2}{5}$ in.) in diameter, and gradually becoming more or less hemorrhagic; and this is followed by a petechial eruption. Sometimes the rash appears in the form of rose spots, which may disappear on pressure. In children the rash may be severe, and resemble that of measles. During the second week the general symptoms are much aggravated. The delirium, as a rule, becomes very intense as the fever reaches the fastigium. Retention of the urine is common. Coma vigil is frequent, in which condition the patient lies with the eyes open, but unconscious. Carphologia is also common.

There is usually an increase in the number of erythrocytes and hemoglobin percentage. There is always a leucocytosis varying from 14,000 to 50,000 per cubic millimeter. In uncomplicated cases there may be an increase of 8 per cent. of the polymorphonuclear cells, and a decrease of the mononuclears and lymphocytes. In severe cases there may be hypostatic congestion of the lungs. The heart also frequently becomes feeble, and there are signs of myocarditis. From the foregoing it will be appreciated that there may be any number of varieties of the disease, varying from very mild cases to those of the more virulent type.

Bronchial pneumonia is the most common complication, and in some epidemics gangrene of the toes, hands or nose may occur; and children frequently suffer with noma or cancrum oris. Paralyses due to postfebrile neuritis are infrequent.

The mortality ranges in different epidemics from 12 to 20 per cent. It is very much lower among the young. After

middle age the mortality rapidly rises, and in some epidemics reaches 50 per cent. Death usually occurs during the second week, and is probably due to toxin. One attack of the disease probably confers permanent immunity.

During an epidemic the *diagnosis* presents few difficulties. Isolated cases, and especially mild cases, such as described by Brill, may be difficult to distinguish from typhoid fever. The positive Widal reaction and blood-cultures in the latter disease will leave no doubt after the first week. The onset is usually with chills, which are rare in typhoid. At times it is most difficult, if not impossible, for the most expert clinician to make a diagnosis. Malignant malaria may simulate typhus. At the termination of the fever cycle the fall by crisis in typhus, and by lysis in typhoid, is an important distinguishing point. The more positive signs are the leucocytosis, associated with the rash, and extreme nervous prostration. Relapsing fever can be readily distinguished from typhus by the spirilla in the blood. Koplik's spots and the coryza will readily exclude measles.

TREATMENT.

In general, the treatment of typhus fever patients is in every respect similar to that of typhoid. Hydrotherapy to keep down the temperature should be regularly employed. This will probably relieve the nervous symptoms as effectively as it does in typhoid fever. It is well to begin the treatment with fractional doses of ¼ grain (0.01 Gm.) of calomel every hour until bowel action has been obtained, and this should be followed by several tablespoonfuls of magnesium sulphate. The heart must be carefully watched, and if it weakens heart stimulants like strychnin or digitalis should be administered. Some authors have used serum taken from cases which have recently had typhus fever, and in those who are very ill it is believed that its intravenous use is a procedure well worth trying. Water should be given freely. For those who have been accustomed to alcohol, small doses seem to be of considerable value. The diet should be liquid until the fall in temperature. The patient should be placed, preferably, in a well-lighted, airy room, or treated on a veranda or in a tent, if climatic conditions permit.

The researches of Nicolle, Anderson and Goldberger, in which the transmission of the disease by body lice was definitely proved, have made it possible to place the prophylactic measures upon a sound scientific basis. The *Pediculus vestimenti* being so definitely concerned in the transmission of the disease, it is quite possible to understand why overcrowded, filthy and unhygienic surroundings are so intimately associated with typhus. The prevention of typhus, therefore, resolves itself upon the eradication of the body louse. This insect does not travel far, except as it may be carried on the bodies of people or in baggage. In all preventive measures it is important not only to destroy the body lice, but also their eggs. The patient's clothes should be removed and thoroughly boiled or treated with steam under pressure. Strong chemical solutions are not to be depended upon to kill the lice and their eggs. The bodies of typhus patients and others who have become infested with lice should be washed with gasolene. This is necessary, owing to the fact that the body lice very frequently bury themselves in the skin, and an oil-removing substance like gasolene or ether is essential to their removal. This should be followed immediately with a hot bath. It is also advisable to use a very strong alkaline soap, and for this purpose the so-called sea-water soap, when used in fresh water, is of considerable advantage. Those who come in contact with typhus patients, especially doctors and nurses, should take all possible precautions against being bitten by body lice. A fairly good prophylaxis can be obtained by using special gowns and boots, the openings of which are tightly closed, especially at the neck, wrists, and tops of the boots. This may be accomplished with some elastic material. Insect powders or solutions are of some value when applied to the body and clothing. Powdered naphthalene is fairly efficient in preventing the lice from entering or living in personal effects. Eucalyptus, camphor, oil of citronella, ordinary kerosene, and powders composed of starch and camphor, are all more or less used. The clothing of all attendants should be frequently changed, and thoroughly boiled before being used again. Whenever effective measures for the destruction of body vermin are carried out, the disease rapidly disappears.

To prevent typhus being carried into a country, all persons from infected regions should be thoroughly bathed, and all their textile effects should be disinfected with steam under pressure in a modern steam chamber. When no steam chamber is available, effects may be boiled or steam used in a tight compartment, as a refrigerator car, for instance.

BUBONIC PLAGUE.

Bubonic plague is an acute, specific, dangerous, communicable disease, caused by the *Bacillus pestis,* and usually spreads to man from rats through fleas; to a slighter extent through droplet infection from persons afflicted with pneumonic plague, or by inoculation. The infection is known also as black death, pestis, mahamari (India), yeki (Japan), and kotawen (China).

It is difficult to state, from a review of the literature, whether the disease described by classic writers was the bubonic plague of the present day. Any disease occurring in epidemic form and causing a large mortality is frequently referred to as plague. The Bible contains references to an epidemic disease among the Philistines which produced glandular swellings in human beings and killed rats. The first more or less reliable accounts of plague come from the second century. In the fourteenth century plague is estimated to have destroyed one-fourth of the population of Europe. The disease occurred in epidemic form in Western Europe until the middle of the seventeenth century. In London, in 1665, it raged virulently, and is said to have been responsible for 70,000 deaths. It disappeared from Eastern Europe in 1884. It made its reappearance in Europe at Oporto in 1899. Since that time small outbreaks have occurred in a number of the principal port cities in Italy, Scotland and England. These outbreaks were traced to ships that had been trading with India and Egypt, where plague appeared in 1899. Until the latter part of the nineteenth century the disease had almost disappeared, but it was presumed that it slumbered in certain parts of China. In 1894 it appeared at Hong Kong, and from there may be said to have spread by maritime routes all over the earth. Hong Kong is one of the great shipping ports of

the world, and as there is very little medical supervision over incoming and outgoing ships, the conditions were ideal for promoting the spread of the disease unhindered. The ports of India are first supposed to have been infected from Hong Kong. Egypt probably became infected in 1898 from India. Japan has become infected a number of times, and the disease still exists there. The infection was probably derived from Hong Kong, and, perhaps, at other times through rats in cargoes from India. The disease was recognized in Manila in 1899; probably a direct importation from Hong Kong. Some places throughout the East, although in active communication with plague-infected centers, remained remarkably free of the disease. Notable instances of this kind are Singapore, Java and Colombo. This freedom is probably due to the lack of docking facilities, which prevented plague rats from having access to the shore. Eventually all of these places became infected, and during the past few years Java has had an outbreak, which has already cost more than a hundred thousand lives. In India the plague mortality goes into the millions. In 1899 the disease had reached South America, and particularly affected the ports of Buenos Ayres and Rio de Janeiro. Madagascar and Mauritius have likewise suffered very severely. By 1900 the disease reached San Francisco from Hong Kong.

Numerous plague conferences, both international and national, were held from time to time, but as the etiology of the disease was not clearly understood, the rules and regulations which were formulated accomplished little. However, apparently few cases occurred in places like San Francisco (U. S.), Glasgow (Scotland), Sydney (Australia), and other countries in which good health departments existed and energetic steps were taken to bring the disease under control. It was not until 1907, at the Second India Plague Commission appointed by the British Government, that the mode of transmission was definitely proved, and prophylactic measures based thereon became available.

Geographically, the disease is not confined to any special latitude or climate. The fact that more cases and greater spread have taken place in the tropics is, in all probability, due

to the poor facilities which exist in these countries for combating the spread of epidemic disease.

The records show that the disease spreads as rapidly under poor hygienic conditions in Siberia and North China as it does in India or other tropic regions.

Yersin and Kitasato,[121] working in Hong Kong in 1894, discovered independently the specific organism of plague. The *Bacillus pestis* may be found in the blood, the swollen glands, the sputum, the organs, especially the spleen, and elsewhere in the human body. The micro-organism usually may be recovered in pure culture from characteristic buboes. It is a short, thick coccobacillus, 1½ to 2 microns in length, and from $\frac{5}{10}$ to $\frac{7}{10}$ in breadth. It has round ends, and resembles the organism of chicken cholera. It is a bipolar staining bacillus, and usually decolorized with the Gram method.

Typical attacks of plague are produced when the bacillus is inoculated in monkeys, cats, rats, guinea-pigs, squirrels, mongooses, bats and marmots. In bovines and equines it only produces local reactions. Canines, birds and reptiles apparently are immune. Plague causes epizoötic among rats, either in acute or chronic form. In the latter condition it probably maintains itself between epidemics. The rat is probably the true reservoir of the disease, and man only its accidental victim. The Indian Plague Commission[122] of 1908 came to the following conclusions: "Contagion occurs in less than 3 per cent. of the cases, playing a very small part in the general spread of the disease. Bubonic plague in man is entirely dependent on the disease in the rat. Infection is conveyed from rat to rat, and from rat to man solely by the means of the rat flea. A case in man is not in itself infectious. A large majority of cases occur singly in houses. When more than one case occurs in a house the attacks are generally simultaneous. Plague is usually conveyed from place to place by imported rat fleas, which are carried by people on their persons or in their baggage. The human agent himself may escape infection. Insanitary conditions have no relation to the occurrence of plague, except in so far as they favor infestation by rats. A non-epidemic season is bridged over by acute plague in the rat accompanied by a few cases in human beings."

Plague does not flourish with equal virulence throughout the different seasons of the year. For instance, for more than twenty years the annual plague curve of Hong Kong has reached its fastigium during May, and its low point during December. This seasonal variation has not been satisfactorily explained. It is usually ascribed to the variations in rat or flea breeding at different times of the year. Observations made in Java showed clearly that during the greatest incidence of plague, the average number of fleas per rat was very much higher than during the period of its lowest incidence.

There is considerable difference in the ability of different species of rats to convey the disease, as well as a considerable percentage of difference in the rats of a given community. The Indian Plague Commission,[123] for instance, found that of 1776 plague-infected rats caught in Bombay, 1334 were *Epimys norvegicus,* and also that during the non-epizoötic period the *E. norvegicus* was the only rat in which plague could be found, notwithstanding this species is not nearly so numerous as the *E. rattus.* It was stated that the *E. norvegicus* usually had about double the number of fleas found on the *E. rattus.* The *E. norvegicus,* however, is more common in compounds, stables, warehouses, and grocery-stores; whereas the *E. rattus* is frequent in sewers, drains and stables, and is seldom found in houses above the third floor. It is especially noted for its burrowing proclivities. Creel[124] showed that in Porto Rico the *E. norvegicus* was the most common, as may be seen from the following tables:

Classification of All Rodents Examined in Porto Rico from June 23 to January 11, 1913.

Norvegicus	23,453	Mongoose	233
Rattus	4,201	Unclassified*	309
Alexandrinus	5,962		
Musculus	5,137	Total number examined	39,295

Classification of Infected Rodents.

Norvegicus	37	Unclassified	24
Rattus	4		—
Alexandrinus	1		66

* The unclassified were those examined during the first week of laboratory examination.

In 68,667 rats caught in Manila during 1912 and 1913 the proportion of the different species was about the same as in Porto Rico. The Indian Plague Commission showed that transmission from rat to rat is by means of the rat flea. Cages were prepared in which infected and healthy rats were only separated by a fine mesh gauze, so as to prevent the transmission of fleas, and no infection took place. In other cages, in which no provision was made for preventing the fleas from transferring themselves from sick rat to healthy rat, infection invariably took place. Healthy rats in cages, hung 4 inches (10.16 cm.) above plague-infected rats, did not contract the disease, although no wire screen was used. This was due to the fact that a flea cannot jump higher than 4 inches (10.16 cm.). When the cages were lowered to within jumping distance of the infected rats, the healthy rats contracted plague. Many variations of this experiment have been carried on by the Indian Plague Commission and other observers, and the work has now been so thoroughly confirmed that practically no doubt exists.

The flea directly concerned in the transmission of plague is the one usually found on rats, namely, the *Pulex cheopis,* although other fleas are believed to be capable of conveying the disease. The exact method by which fleas transmit plague has not yet been definitely established. It was assumed at first that in the act of biting the human host the plague flea injected plague organisms. Other observers were of the opinion that infection was conveyed through defecation of the flea at the time it bit its victim, and the itching caused by the flea-bite afforded an opportunity for the organisms to be rubbed into the skin. More recent work done at the Lister Institute has shown that fleas usually regurgitate plague organisms, and in this way the skin becomes infected.

The Indian Plague Commission believed the spread of plague was due to fleas being conveyed in merchandise or on human beings, rather than by the migration of rats. This conclusion is open to considerable doubt, because plague fleas removed from their host are very short-lived, probably not more than a few days, and even a shorter time under unfavorable conditions such as sunlight. Experience in Manila showed that practically every case of plague could be identi-

fied more or less directly with fleas that were not far removed from a plague rat. But this direct connection only became apparent after considerable experience in finding rats. It is generally found that a dead plague rat could be found in a ceiling with cracks, or a floor with openings, or in a hollow wall close to a bed in which the human victim slept. It is assumed that the plague flea, not finding another rat for its host, sought its human victim. It was also possible in Manila to prove that fleas were responsible for the transmission of the disease, by placing healthy guinea-pigs in the bed or room in which the human victim died. In the course of four or five hours, rat fleas would be found on the guinea-pigs. If permitted to remain, the guinea-pigs often died of plague.

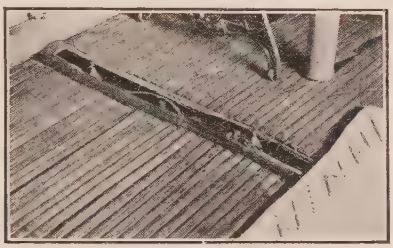


Fig. 15.—Rat shown in bamboo joint of the floor.

There is much reason to believe that so long as fleas can find rat hosts they will not attack human beings.

In tropical countries bamboo structures have been found to be directly associated with the spread of the disease (see illustration). This is due to the fact that the round bamboo joints make admirable harboring places for rats, and when rats die of plague the fleas leave these hiding places, and frequently find a human victim. In some sections of Manila, for instance, and in many parts of Java, plague was eradicated by cementing the ends of bamboos used in construction.

Pneumonic plague probably spreads directly from man to man. For instance, in the outbreak in Manchuria many doctors and nurses were victims of the disease, but as soon as proper masks were used by those caring for the sick the infection among attendants ceased. Strong and Teague found plague bacilli in droplets of mucus expelled during coughing,

or sometimes even during speaking. Pneumonic plague is more likely to spread in cold countries than in warm countries. This is probably entirely due to lack of air dilution. It is obvious that a case of pneumonic plague in a house with closed windows and doors and inadequate ventilation, such as are the rule in cold countries, would be more liable to surcharge the air with plague bacilli. Whereas in tropical countries, where doors and windows are kept open, the dilution of the air due to better ventilation would greatly diminish the danger of spread.

There is no particular difference with regard to the sex incidence of plague, except in so far as persons are likely to be exposed to rats or humans who are suffering with pneumonic plague. In all probability, pneumonic plague has its origin through the secondary invasion of the lung which occurs in bubonic cases. When the lungs are involved, especially in cases located in poor hygienic conditions, its transmission directly by droplet infection from human to human is quite conceivable, and may be the origin of a pneumonic outbreak.

In California a more or less permanent reservoir for plague exists in ground-squirrels. Elaborate measures have been taken to free large farms of squirrels, and, on account of the extensive work done, transmission to human beings seldom takes place. It is stated by some authors that plague may be found in marmots, and by others in the tarabagan. The great China epidemic of 1911 is said to have begun among trappers of these animals. The intense cold caused much overcrowding in poorly ventilated quarters, with the result that the pneumonic type soon became predominant. Large numbers of laborers soon afterward proceeded from North China to South China by railway. As it is not customary for trains to run at night in China, the nights were spent in badly ventilated, overcrowded lodging-houses. The disease, once introduced among these passengers, spread with alarming rapidity. As these laborers proceeded to many provinces in South China, they became the centers of infection, and a widespread epidemic occurred.

Outbreaks of human plague are almost invariably associated with considerable mortality among rats. In communi-

ties which have a modern health service it is customary to make periodic examinations of rats, in order to determine whether plague exists among them. If it is found, adequate steps are at once taken to exterminate rats and bring the disease under control. In this way human outbreaks have been reduced to small proportions, and not infrequently entirely prevented.

The great majority of human plague cases originate from the bites of fleas that have been infected by biting rats suffering with the septicemic type of the disease. At rare intervals it is possible to find the probable site of the entrance infection, and plague bacilli may be recovered from the skin and underlying tissues involved in the wound produced by the bite. The bacilli, after being introduced, travel by way of the lymphatics to the nearest lymphatic glands. The resisting power of the glands is almost invariably overcome, and the infection then spreads to the thoracic duct, enters the blood-stream, and causes septicemia. Another mode of entrance into the blood is probably through the direct absorption caused by the degeneration of the cells in the lymphatic glands. The glands most usually affected are the femoral, the inguinal, the axillary and the cervical. It is noteworthy, however, that the fact that most cases of plague implicate the glands of the groin does not necessarily mean that the infection has entered through the lower extremities. In cases of experimental human plague in Manila the infection was introduced by a hypodermic needle in the arm in the vicinity of the deltoid, and in the majority of cases the first physical signs of the disease were the characteristic bubonic swellings of the inguinal region. From the primary bubo the infection may travel up the lymphatics, and produce secondary buboes in other chains of glands. In other instances it is also quite possible that the bacilli may gain direct entrance to the blood-vessels through injury to the walls of the veins in primary buboes.

In the pneumonic variety transmission undoubtedly takes place by direct infection through the respiratory or alimentary tract. Strong[125] and his co-workers, Teague and Crowell,[126] show that the primary infection in pneumonic cases was apparently in the bronchi, and by extension into the

lung-tissues. According to Simpson,[127] infection also probably takes place through the intestinal tract by the consumption of plague-infected food. But at best this must be a very rare way for the disease to be transmitted. Blood-cultures made during the febrile stage of the disease are almost always positive for the plague bacillus.

The most characteristic pathologic feature in connection with plague autopsies is the bubo, of which over 50 per cent. are found, primarily in the femoral region. There is considerable suggillation in the dependent portions of the body, which has the appearance of large black spots. It is this sign which led to the name "black death." It is also quite common to find very small hemorrhages into the skin which are frequently not found on the autopsy table, unless their presence has been noted in the living case. At times there are very small nodules about a millimeter ($\frac{1}{25}$ in.) in size which, upon incision, exude a turbid fluid which contains plague bacilli. The primary bubo is found in the lymph-glands which drain the area of the skin that forms the portal of entry of the plague bacillus. The glandular mass feels boggy and elastic. The individual glands cannot be readily palpated. On section these buboes are found to be very edematous, and exude a large amount of yellowish fluid. The appearance of a plague bubo is very characteristic. A large amount of the fluid exudes as soon as the skin over it is incised. Its edematous character distinguishes it readily from buboes due to other infections. Plague bacilli disappear from the glands usually about the time pus-formation starts. The lesion in the gland is a hemorrhagic inflammation and coagulation necrosis. Other glands located along the lymph-channels are also involved. In severe septicemic cases all of the glands may be enlarged and swollen, but large buboes are uncommon. Secondary plague lesions occur in the lungs, but these may be distinguished from primary pneumonic cases. The lesions in the lungs may be bronchopneumonic in character. There may be peripheral infarcts, and the metastatic type of infection. There may be punctiform hemorrhages of the pleura.

The spleen is usually enlarged. The capsule is tense, opaque, and varies in color from a reddish to a bluish appear-

ance. The capsule is often studded with small, confluent hemorrhages. On section it does not collapse nor lose its form. Its consistence is firm. The cut surface is grayish red in color and rather dull in appearance. There are usually small infarcts and necrotic nodules. Cultures in the spleen are usually positive. The liver shows changes found in acute parenchymatous inflammation.

The changes in the kidney can scarcely be said to be characteristic, and are those usually found in acute febrile conditions. There is often degeneration of the tubular epithelium. The heart nearly always shows parenchymatous or fatty degeneration in the myocardium. The endocardium, as well as the myocardium, at times shows small hemorrhages. The most common lesion is a small hemorrhage in the epicardium.

The *incubation period* of plague varies from two to ten days. In most cases it occurs within a period of three days. For clinical purposes the disease may be divided into four types: pestis minor, bubonic, septicemic plague and pneumonic plague.

Pestis Minor. In this type of plague the patient has an irregular fever, swelling in the glands of the groin, and possibly suppuration. Very often the patient is not ill enough to seek relief. These cases are commonest at the beginning and end of epidemics. Plague bacilli can usually be found in the glands, and in the blood, if sufficiently large quantities are taken during the height of the fever. Diagnosis can often be arrived at much easier by searching for the typical plague vesicle, which probably denotes the entrance of the infection, and finding therein the plague bacilli.

Bubonic Plague. There may be prodromal symptoms, consisting of general malaise, headache, pain in the back, uneasiness, chills, and mental apathy. This variety occurs in probably three-fourths of all the cases. The onset is sudden, with a rise in temperature to 103° or 104° F. (39.4° or 40° C.), with a corresponding increase of the pulse and respiration rate. There is usually a marked increase in the prodromal symptoms. The mental dullness is very characteristic, and often leads to suspecting the disease. The patient's expression is that of fear and anxiety. His eyes are bloodshot, bright and staring. The face is drawn, and often the

nostrils are dilated. The temperature may remit somewhat on the second day, but usually rises again almost immediately afterward. If recovery takes place, the fall of temperature is usually by lysis. In fatal cases the temperature often falls rapidly to normal or subnormal, and then rises quickly to 107° F. (41.6° C.), followed by death. Glandular swellings become prominent on the second day, and very often can be felt by careful palpation on the first day. If the patient survives until the fourth day the swellings are usually quite large and distinct. Suppuration may occur, or, in some instances, gangrene. Suppuration is usually not regarded as a favorable symptom. Petechiæ usually appear about the third day, and are often referred to as the so-called "black spots" or "tokens of death," and gave to the disease in the Middle Ages the name of "black death." Hemoptysis frequently occurs. Plague bacilli can usually be found during the high fever periods of the disease. There is a leucocytosis from 90,000 to 100,000, and the red cells and the hemoglobin are distinctly increased. The increase in the white count is due almost entirely to the polymorphonuclear leucocytes. The breathing is rapid, the breath sounds are harsh, and there are generally moist râles.

The urine is usually diminished in amount, and contains albumin and casts. Pregnant women generally abort. There is low muttering delirium, which gradually passes into coma. Death usually occurs between the third and fifth days. In favorable cases the tongue becomes moist, the pulse rate and temperature fall, and delirium gradually abates. Even in favorable cases buboes generally continue to enlarge and soften. If not incised, they usually burst spontaneously. As a rule, they are very ill-smelling. In these cases convalescence usually begins between the sixth and tenth days.

Septicemic Plague. In this form of the disease the patient usually succumbs within three or four days, and before the appearance of buboes. The symptoms are similar to those of any general septicemic process. The degree of virulence and rapid course of the disease depend on the entry of large numbers of bacilli into the blood. The patient is extremely prostrated from the beginning. He is pale and apathetic, and, as a rule, there is no great febrile reaction. The temperature

may not be above 100° F. (37.7° C.). Vomiting is severe, and diarrhea with blood frequently occurs. There are often petechial hemorrhages in the skin. Leucopenia is the rule in these cases. Briefly, there is an overwhelming infection in which the patient succumbs before the defensive forces of the human organism have had an opportunity to act.

Pneumonic Plague. This variety of plague must be carefully distinguished from the ordinary inflammation of the lungs found secondary to bubonic plague. This is a true pneumonic plague, and begins suddenly with fever, shortness of breath, coughing, and frequently pain in the chest. There is expectoration of bloody mucus, which contains plague bacilli. Cyanosis comes on early. The pulse is small and rapid. There is early enlargement of the spleen, and death usually occurs within four days. Recovery in this type of the disease is very rare. The other symptoms correspond very closely to those of pneumonia caused by the pneumococcus or other organisms.

Diagnostic errors in plague may have grave consequences, and the first cases in a community in which plague is rare, or has not heretofore appeared, are very likely to be overlooked. But once the presence of the disease is suspected, its *diagnosis* is comparatively simple, and can be made practically certain by resort to laboratory methods. Very often the disease is mistaken for typhoid fever; but the appearance of the buboes, and finally the demonstration of the plague organisms, make the distinction certain. The pneumonic type is very likely to be overlooked, and very often only attracts attention through nurses and doctors becoming affected with the disease. In the tropics plague is often confused with ordinary glandular fever, and, as a rule, many cases are sent to plague hospitals by mistake. The clinical examination alone usually will suffice to distinguish the disease with reasonable certainty. In glandular fever the glands are exceptionaally hard and movable, whereas the plague bubo is more in the nature of an edematous mass, in which it is difficult to palpate the individual glands. There is very little prostration in glandular fever, whereas in plague prostration, as a rule, it is very great. Bacteriologic diagnosis based upon material taken from the gland, or by direct examination of the blood may be

depended upon to show plague. Films can be made of fluid drawn from the suspected bubo and stained with methylene blue, which has been diluted with carbol-fuchsin. The presence of the typical bipolar staining bacilli is almost sufficient evidence upon which to make conclusive diagnosis. An absolute diagnosis must be dependent upon blood-cultures and animal experiments. In this way diagnoses can be confirmed by inoculating rats or guinea-pigs with blood or material taken from a bubo, and, upon the test animal becoming sick, further inoculations from that animal into a healthy animal, which should cause the disease.

The mortality from plague is probably higher than that from any other epidemic disease which occurs in considerable numbers. As a rule, the mortality varies from 80 to 90 per cent., and many outbreaks have had a mortality of 95 to 98 per cent. The prognosis depends very largely on the character of the outbreak,—that is, upon the virulence of the strain of plague bacilli. In the pneumonic form of the disease the recorded mortality in reliably diagnosed cases is 100 per cent. The septicemic type has a higher mortality than the bubonic variety.

TREATMENT.

There is no specific for plague. Many serums have been made from time to time, but none of these can be said to have any appreciable influence on the mortality of the disease, unless they have been given during the prodromal or incubation period. The treatment resolves itself largely into making the patient comfortable and following the same general principles which apply to the care of fever patients. There should be purgation and stimulation, with the use of morphin to control the pain. It is customary to apply ice compresses to the buboes. With regard to the use of serum, while there is no satisfactory evidence that it is of value, there is considerable evidence that it never does any harm, and it may be of some service. Fever may be controlled by sponging and cold applications. If the buboes break down they should be incised and have antiseptic treatment. Ichthyol is frequently recommended for this purpose. The heart should be sustained by digitalis and strychnin. There should be administration

of sufficient fluid to keep the kidneys active. The diet should be of the liquid variety. Milk is usually well borne.

Prophylactic measures for the control of plague depend upon preventing rat fleas that have bitten plague-infected rats or human beings from biting humans. To accomplish this, means the killing of rats or removing them to places remote from human contact. The spread of human plague is probably greatly retarded by the fact that rat fleas do not bite man by choice, but only after failure to gain lodgment on rats. It thus happens that many persons contract the disease who sleep in the neighborhood of places infested with rats. In countries in which bamboo enters into the construction of houses, and particularly beds and furniture, plague is often indirectly spread through this means. Rats are very fond of nesting in bamboo-joints, and when they happen to die of plague and the fleas have no other rat to which they can go, they seek other food supplies, and very often find persons who sleep, for instance, in beds made of bamboo or in the vicinity of bamboo structures. Hollow walls in which rats die are also frequently directly concerned in transmitting plague to persons. Likewise persons who sleep on floors below which there are hollow ceilings, which afford harborages for rats, are very frequently infected. Persons who go about barefooted and barelimbed, in the vicinity of rat nests which have been inhabited by plague rats, are very liable to infection. It is quite noteworthy that for years it was known that persons who worked in oil warehouses or soiled their clothing with kerosene very seldom contracted the disease. The reason for this, of course, is now quite obvious. Fleas dislike the smell of kerosene very much, and will give it a wide berth.

From the foregoing it will be apparent that the prophylaxis readily divides itself into two classes: public and personal.

Public Prophylaxis. This consists in obliterating rat-breeding places in the vicinity of man's habitations. This is usually brought about by preventing the construction of hollow walls, hollow ceilings, posts, etc. Rats are naturally secretive animals, and if their means of hiding are taken away they are inclined to leave human habitations. In most coun-

tries, especially in port cities, in which plague is liable to prevail, ordinances are being gradually adopted which prevent the construction of buildings which are not reasonably rat-proof. This also applies to docks and piers, so that rats coming from ships that have touched at plague-infected ports may not find harborage on the piers. During the presence of an outbreak of plague, the primary measures for combating the disease consist in destroying the rats, and preventing their harborage near man. For instance, if the problem concerns rat control in a city, the method employed by the author in Manila is probably the most successful:

A list of the places at which plague-infected rats were found was made. Each was regarded as a center of infection. Radiating lines, usually five in number, were prolonged from this center, evenly spaced like the spokes of a wheel. Rats were caught along these lines and examined. Plague rats were seldom found more than a few blocks away. The furthermost points at which infected rats were found were then connected with a line. The space inclosed was regarded as the section of infection. The entire rat-catching force, which had heretofore been employed throughout the city, was then concentrated along the border of the infected section. They then commenced to move toward the center, catching the rats as they closed in. Behind them thorough rat-proofing was carried out. One section after another was treated in this way until they had all been wiped out. Once weekly thereafter rats were caught in the previously infected sections, and at other places which were insanitary, and which had been infected in years gone by. This continued for one year.

Methods of Destroying Rats. Rat trapping or poisoning is usually employed. The relative effectiveness of various rat-traps shows that a wire spring or snap trap has an efficiency of 7.47 as against 0.97 for the wire-cage trap, and 0.12 for poisoned bait. Many different forms of rat poisons are advocated. The one found most successful in Manila consisted in boiling 1 part white arsenic with 4 parts, by weight, of rice, and distributing this about in places frequented by rats. One grain of the rice is sufficient to kill a rat. Recently (1917) researches at the Kasauli Institute indicate that barium carbonate is very efficient. In seaports precautions must be

taken to prevent infected rats from gaining access to vessels, and also to prevent rats from other ports from gaining access to the shore. This is best accomplished by thorough fumigation of such ships at frequent intervals. This is best done usually with a 2 per cent. sulphur-dioxid gas. Carbon monoxid and carbon dioxid have been recommended from time to time, but are not regarded as satisfactory, because they only kill the rat and the flea escapes. An apparatus devised by Harker consists in using flue gases from steamers or launches. This has proved very successful in killing rats, but failed to kill fleas. Probably the most successful agent is hydrocyanic gas, but it is so extremely dangerous that many fatalities have already been recorded from its use.

Taking everything into consideration, the best method for killing rats on board ships is probably by using sulphur dioxid. This can be readily generated in iron pots that have been set into tubs of water. These are lighted, the places tightly closed, and the gas allowed to remain for a period of at least six hours. Two pounds of roll sulphur for each 1000 cubic feet of space are sufficient. Rat-guards should be used on the lines by which vessels are tied to wharves, in order to prevent rats from passing back and forth. At night the gangway should be lifted. Vessels from plague-infected ports should also be fended away from the wharf for a distance of at least three feet.

The rat virus, which has been recommended from time to time for the purpose of spreading fatal disease among rats, has proved very disappointing. It soon loses its efficiency, and when it does act, often obscures the diagnosis of plague among rats.

In connection with the foregoing measures, provision must be made for isolating human cases, and adequate precautions taken to prevent fleas with which they may have been infested from gaining access to healthy individuals. It is also deemed advisable to disinfect the sputa or excreta which come from the plague sick. A bacteriologic laboratory is an absolute essential in dealing intelligently with plague outbreaks, where diagnosis may be reliably and satisfactorily made.

Personal Prophylaxis. This consists of avoiding infected regions and guarding against flea-bites. For those who are

compelled to enter plague-infected areas some measures of precaution may be obtained by sprinkling kerosene or some other insecticide about the tops of the shoes, armlets, and neck-bands. A much more reliable prophylaxis, however, is found in plague vaccine. Hafkine's and other vaccines give protection for a period of at least six months and perhaps longer, and those who are constantly exposed to plague fleas should protect themselves in this way. So-called true vaccines—that is, those made with attenuated living plague bacilli—are generally held to be more effective than those made with the dead bacilli, as is done with the Hafkine method. Those who come in contact with pneumonic plague should wear head-masks which may be made of Canton flannel, with suitable eyes of celluloid.

After plague has disappeared from a city it is well that examinations should be made at weekly intervals of a limited number of rats, particularly those in areas in which plague infection has prevailed. Effective work done in this direction and adequate measures taken when plague-infected rats are found will probably prevent outbreaks among human beings.

FILARIASIS.

Filariasis, also known as filarial disease, is an infection of man by any species of filaria, and is transmitted through the bite of a mosquito that contains microfilaria obtained from an infected man or animal. The disease is characterized by various manifestations, elephantiasis, or swelling of the lower limbs, being the most prominent.

Reliable knowledge with regard to this disease begins in 1863, when Demarquay[128] discovered in the tunica vaginalis a larval nematode in a case of chylous dropsy, which has since been named the *Microfilaria bancrofti*. The huge legs in some cases of filariasis are so striking that they were noticed by ancient writers, who gave descriptions which clearly refer to elephantiasis. Chyluria was described in very early times. In 1812, for instance, Chapotin[129] described this condition among the natives of Mauritius. Wucherer[130] found filaria in 1866 in the urine of persons afflicted with chyluria. Lewis[131] made a similar observation in 1870, and in 1872 discovered that the blood of man was the normal habitat of this larvail parasite, and named it

Filaria sanguinis hominis. In 1876 Bancroft,[132] in Brisbane, Australia, discovered the adult worm. Cobbold[133] named it *Filaria bancrofti.* In later years the subject has aroused increasing interest, and the disease is now known to be an enemy of man throughout the tropical world. According to Manson,[134] the human circulation is the habitat of the larvæ of at least five distinct species of filaria. Only one parasite, namely, the *Filaria bancrofti,* appears to have any important pathogenic significance. The *Filaria loa* may also be concerned.

Filariasis has been found throughout the tropical and subtropical world; in Europe, as far north as Spain; in America, at Charleston, S. C.; on the other side of the Equator, as far south as Australia. In certain parts of China 10 per cent. of the population are said to harbor it. In some countries over 50 per cent. of the natives have microfilaria in their blood. According to Bahr,[135] 27.1 per cent. of the Fijians harbor the microfilaria in their blood. Bahr also states that at one time or another nearly every Fijian is subject to filariasis, and concludes that with 27.1 per cent. in whom it is possible to demonstrate microfilaria, added to 25.4 per cent. having symptoms of filariasis without its being possible to demonstrate the microfilaria, there is an infection rate of 52.5 per cent. of the entire population. Heavy infections have also been found in the Friendly Islands, Samoa, Madras, Demerara, the West Indies, the Philippines, and West Africa. It is more than likely that a careful research would show the presence of the disease in practically all tropical countries.

In 1877 Cobbold[136] showed that the microfilaria was introduced into the body by the bite of the mosquito. Soon afterward Manson,[137] working in Amoy, proved the transmission of the disease by the mosquito. According to Theobald,[138] the known mosquito-carriers of the worm are the *Culex fatigans, Mansonia uniformis, M. pseudotitillans, Pyretophorus costalis, Myzomia rossi, Myzorhynchus nigerrimus, M. minutus, Cellia albimana, Stegomyia pseudoscutellaris.* According to Bahr,[139] the worm is capable of development in the *Culex faitgans,* but the favorite intermediary in the Fiji Islands is the *Stegomyia pseudoscutellaris.* The *Culex jepsoni* may also be involved. It is not understood why the filariæ will not go on to full development in the different mosquitoes. The filaria develops in the thoracic muscles

of the mosquito and then passes to the salivary system and is injected in the same way as the malarial parasite. If placed on the skin, however, it may work its own way into the body, similar to that of the ancylostoma embryo. After the organism once penetrates the skin its further history is not known until it reaches the adult condition. A male and a female adult are generally found lying together. Females usually predominate in numbers. Dead and calcified worms have been found in the lymphatic glands, testes, epididymis, spermatic cord, and tunica vaginalis. In these locations the female produces the microfilariæ which pass through the lymphatic glands and thoracic duct into the blood-stream, in which they are usually found at night, and only under rare conditions in the daytime. Bahr, in the Fiji Islands, showed that in that country this nocturnal periodicity was not maintained. He found them constantly in night and day blood. The adults lying in the lymph-channels may cause obstruction to the lymph, and this produces varices and inflammation of the glands, which may result in the extravasation of lymph or chyle. It may be said that, in regions in which there is no filariasis, elephantiasis is very rare, and it is doubtful whether the few cases that are found are contracted in those communities. Adult filariæ are sometimes found in the tissues removed during operations for elephantiasis. The disease elephantiasis is probably produced by the attacks of lymphangitis which result in blocking the lymph-channels. It has been alleged that bacterial infection may be responsible for these inflammations, and Dufogere has described a diplococcus which he believes is responsible. The probable cause of elephantiasis in the tropics is the *Filaria bancrofti,* although it is quite possible that other filariæ may be concerned.

It is generally held that unless the adult worms lie in a position to obstruct the flow of lymph, or that there is injury to the adult female which causes abortion and liberation of eggs which, owing to their oval shape, may block the channels of the smaller lymph-vessels, there are no pathologic effects. The fully developed microfilariæ which circulate in the blood apparently have no pathogenic properties. Two types of filarial disease may result when filariasis causes blocking of the lymph-trunks, namely, one characterized by varicosity of the lymphatics, and the other by edema. The manner in which the filariæ cause disturbance can-

not be stated with certainty. It seems likely that one or more worms may at times obstruct the thoracic duct and act as an embolus or be responsible for the production of a thrombus. The worm may also give rise to inflammatory thickening of the walls of the lymph-vessel which results in obstruction from the resulting stenosis or thrombosis. The general result is a blocking of the lymphatic areas drained by the implicated vessels, with a resulting varicosity or edema, or both.

In lymphatic varix a compensatory lymphatic circulation is soon established, but naturally before this can be effected a rise of lymph-pressure and dilution of the lymphatics in the implicated area must take place, and it is this condition that results in lymphatic varix of different degrees. When the thoracic duct is obstructed a retrograde circulation must take place, and the fluid is forced in a backward direction to the abdominal and pelvic lymphatics. As a result of this action the thoracic duct up to the seat of the obstruction becomes enormously dilated, sometimes to 1½ centimeters (⅝ in.) in diameter. This often leads to the formation of enormous varix, which may be 25 centimeters (9.84 in.) or more in diameter, 5 to 15 centimeters (1.96 to 5.9 in.) in thickness, and conceal the kidneys, the bladder, and spermatic cords. A white or pinkish fluid exudes when this mass is pricked. If the obstruction is below the lacteals the fluid is clear like ordinary lymph. When the varix affects the integuments of the scrotum, a condition called lymph-scrotum results. When the tunica vaginalis ruptures, for instance, there is chylous dropsy of that sac, or chylocele. It will be readily appreciated that a number of different lesions depending upon the site of the obstruction may result.

When filarial disease is associated with lymphatic varix, microfilariæ can generally be found in the circulating blood, as well as in the contents of the dilated vessels. It is only in cases of long standing that microfilariæ are absent at times. Bahr[140] and Manson[141] both state that they have observed cases in which the microfilariæ gradually disappear. Disappearance is attributed to the death of the parent parasites. It is not always possible at autopsy to find the adult worm, and at no time have there been any considerable numbers found.

Sometimes the blocking is due to a coiled mass of worms. Young[142] found six females and one male in a mass. The

obstruction in the lymph-channels may be due to the inflammatory changes caused by the constant irritation of worms or their products. Inflammatory tissue may become organized, and thus the blocking would continue even after the adult worm had been eliminated. Bahr[143] believes that the periodic discharge of the microfilariæ may be a factor in the cause of lymphangitis, orchitis and funiculitis. In elephantiasis arabum the microfilariæ usually are not found in the blood. In cases in which microfilariæ are found they are probably traceable to a fresh infection by the mosquito. The consensus of opinion appears to be that elephantiasis arises by damage to the female worm, which causes her to produce immature embryos. These cause blocking of the lymph-channels, but elephantiasis will not result unless there is infection with microbes in the blocked area. Mechanical blocking alone may cause disturbances like lymph-scrotum, but not elephantiasis. The latter condition sometimes follows operations for the removal of edematous masses like lymph-scrotum.

The miniature egg is 50 microns in length by 34 in breadth, while the fully developed microfilaria is 250 to 300 microns in length and 7 to 8 in breadth.

The lymphatic vessels in the areas involved will be found enlarged and inflamed, and abscesses of varying size may contain dead worms or the *débris*. The worms are often calcified by the deposition of lamellar plates of calcium carbonate. Some of these abscesses on incision contain material similar in appearance and consistency to apple-butter. This substance is found to consist largely of eosinophile cells. Abscesses are most frequently found on the lower extremities. The seats of election are in the gastrocnemius near the condyles and Scarpa's triangle. They are probably due to necrosis caused by the pressure of the worms. The vessel-walls undergo various pathologic changes, In some there is proliferation of the endothelium and invasion of the vessel-wall with fibrous tissue. The adult worm is often found in inflammatory masses adhering to the skin in various parts of the body. A favorite seat is just below the supraorbital region.

A lymphatic varix generally forms part of a larger dilatation of the pelvic lymph-vessels and glands. The glands are frequently perforated with dilated channels, and the vessels leading to them are also enormously enlarged and thickened. On section the glands may present a sieve-like appearance. In chylous extra-

vasations the thoracic duct is frequently found impervious, and the lacteals, in the lumbar, pelvic, and pudendal regions are always enormously thickened. In cases in which there has been chyluria it is sometimes possible to trace the openings of the chylous vessels into the bladder.

CLINICAL ENTITIES.

The various clinical entities which may be caused by filarial infections will be taken up in alphabetical order.

Abscess. Many writers on tropical diseases have drawn attention to the frequency with which filarial abscesses are found in various parts of the body. At times the parent worm dies of unknown causes. As a rule, the dead body of such adults is absorbed just as any absorbable foreign body in the human organism is likely to be. At times the dead worm acts as an irritant, and causes an abscess, in the contents of which fragments of the adult filaria may be found. These abscesses frequently open spontaneously, or they may be opened by surgical means, and usually heal without further trouble. If they should form in the thorax or peritoneal cavity, serious consequences, and often death, may result. Manson is of the opinion that in certain instances abscesses may form independently of the death of the parasite. That is, for instance, in the varicose glands, in lymph-scrotum, and in elephantiasis. The direct connection of abscesses with filarial disease is frequently overlooked. The exciting cause is generally due to the death of the adult worm which causes necrosis due to pressure in cutting off the blood-supply, and thus making such tissues susceptible to infection by ordinary pus organisms. The material in the abscess is, as a rule, very characteristic, resembling apple-butter.

Chyluria. Chyluria is a name given to that condition which exists when chyle is passed with the urine. It is due to the rupture of a chyle lymphatic into the bladder or urinary tract. The rupture is usually caused by filarial obstruction in the thoracic duct, this causing pressure symptoms in the weaker lymph-channels farther down. The onset of the disease is usually sudden, and generally accompanied by pain in the back, and by aching sensations about the pelvis and groins. These symptoms are probably due to the great dis-

tention previous to the rupture of the lymph-vessel. As a rule, considerable amelioration of the pain occurs after the rupture actually takes place. Soon afterward the patient notices that he is passing milky urine, which may be colorless, or range in tint from pink or even red. Sometimes it is quite white in the morning, and reddish in color later in the day. The urine is much influenced by the kind of food taken, and the intervals at which it is taken. At times there is a temporary cessation of chyluria, probably due to a temporary closing of the lymph-vessel.

Chylous Urine. Retention of urine, which frequently occurs, is due to coagulation in the bladder. As a rule, it is quite painful, but does not persist more than a few hours, after which worm-like clots are passed. On standing, a cream-like substance resembling fat accumulates on the surface. Fat-globules are seldom found on microscopic examination. Microfilariæ, white and red cells, and crystals of calcium oxalate may be seen. The specific gravity varies from 1015 to 1020. The reaction is usually acid. If the urine is treated with ether and the fat-like substance removed, it will be found to vary from ½ to 3½ per cent. After the removal of the fat, tests for albumin usually show it to be present. When the quantity of fat is very small the condition is often referred to as lymphuria, and if blood is present it is referred to as hematolymphuria. There may be various combinations of these different urines at different times. Chyluria is not directly dangerous to life, but if it persists over a long period there may be pronounced anemia, depression and debility. As a rule, the patient, under these circumstances, becomes incapacitated for active vigorous life. Chyluria frequently occurs after childbirth. The disturbance of the pelvic lymphatics due to the muscular efforts during labor probably causes rupture of the lymphatics. In men violent exercise, particularly that which brings the abdominal muscles into play, as, for instance, hauling a rope, or leaping, also brings on at times attacks of chyluria in those who are affected with filaria.

Treatment. Various forms of treatment have been advocated, but none of them can be said to be very satisfactory. Disappearance of chyle is no doubt sometimes attributed to

drugs which happen to be given at times when the vessels would have closed themselves without treatment. A favorite treatment is to place the patient in bed, elevate the pelvis, and restrict the amount of food and liquid. A few days' treatment along these lines often results in a temporary cessation of the chyluria. Gallic acid in large doses, benzoic acid, methylene blue, tincture of the chlorid of iron, a concoction of Mangrove bark, chromic acid, glycerin, and ichythol may be given. It would seem desirable to try syrup of ipecac in ½- to 1- teaspoonful (2 to 4 mils) doses. Some cases may lend themselves to operative relief, but in general this method of treatment is not to be commended.

Chylous Dropsies. There may be chylous dropsy into various regions. The seats of election are usually the tunica vaginalis and the peritoneum. In chylous tunica vaginalis there is a filling up of the tissues with an opaque fluid which, on tapping, is found to be chylous, and may contain microfilariæ. Sometimes this condition is preceded by an attack of fever and orchitis. There may be a rupture of lymph-vessels directly into the peritoneal cavity. All of the foregoing conditions, however, except that of chyluria, are apparently rare. The treatment is the same as that for chyluria.

Lymph Scrotum. The scrotum may undergo various degrees of enlargement. A careful palpation will reveal a number of varices, and when pricked with a pin they discharge large quantities of milky fluid. The vessels are filled with chyle. Frequently when filled with lymph the fluid is straw-like in color. Some of these punctures flow until 200 to 250 mils (6.6 to 8.3 f℥) of fluid have escaped. Such punctures often run for many hours, much to the annoyance of the patient by soiling the clothing and by physical exhaustion. Microfilariæ can usually be detected on microscopic examination in such fluid. This condition usually precedes true elephantiasis.

Treatment. The treatment is purely symptomatic. It should consist of mechanical devices for suspending the scrotum, and the use of powder to prevent friction. Surgical intervention is at times indicated, and consists in the excision of the diseased tissue. Great care should be exercised to push the testicles well out of the way during such operations.

Most rigid antiseptic precautions are indicated, owing to the likelihood of infection. When the operation is done under good conditions, the wound usually heals rapidly by first intention.

Chylous and Lymph Diarrhea. Just as there may be microfilaria, it is possible for some of the lacteals or lymph-vessels to establish a connection with the intestinal tract, and chyle or lymph, under such circumstances, may be found in the intestinal discharges. This condition, however, is very rare.

Other Forms of Filarial Disease. From the foregoing description of the pathology of filarial disease, it will be apparent that blocking of the lymph-channels may give rise to lesions in many parts of the body. The location of varices may simulate tumors of various kinds. Large masses are frequently found in different parts of the abdominal cavity. Sometimes their true nature is not discovered until at the time of the operation. Enlargement of the glands of the groin often resembles hernia. At times there is invasion of the testicle with symptoms of orchitis. Hydrocele also occurs frequently.

Treatment. No specific treatment is available. Unless there is discomfort or mechanical interference with physical freedom, varices had better be left alone. After all, they carry on a collateral circulation which may be of service.

FILARIAL LYMPHANGITIS.

Filarial lymphangitis is an inflammation of the lymph-vessels and glands which resembles elephantiasis, but in which the filariæ cannot be demonstrated. Elephantoid disease or elephantoid fever is another term for the affection.

Inflammation of the lymph-glands and vessels occurs in all types of filarial lymphangitis, and is a preliminary condition to elephantiasis, lymph-scrotum, and similar manifestations. At the beginning of an attack there is usually painful cord-like swelling of the lymphatic trunks and associated glands. The attack usually begins with a rise of temperature, varying from 38.5° to 40° C. (101° to 104° F.), often accompanied by vomiting and headache. At times there is

only a red edematous area of the skin, and the glands are not inflamed or painful. In the course of a few days the temperature usually drops to normal, but the erysipelatous rash persists for a number of additional days. The attack usually ends with profuse sweating. The swelling gradually subsides, but never quite reaches normal. Sometimes the tension in the inflamed tissues relieves itself by discharge. If there is an extensive abdominal varix, for instance, there may be symptoms resembling peritonitis, and the case may end in death. The attacks recur at varying intervals, and with the gradual increase of tissue a condition similar to elephantiasis finally results. In Manila the disease was of frequent occurrence, even among the better classes. Among an American population of several thousand, 6 cases are known to have occurred, but in none of them could filariæ be demonstrated.

The true nature of the disease is best shown by the repeated attacks which occur at intervals of weeks, months, or years. The redness and swelling caused by the stings of certain insects may be mistaken for a preliminary attack. In stings it is usually possible to find the primary lesion. Sometimes, owing to the chills and the profuse sweating, the condition is mistaken for malarial fever, but blood examination would show malarial parasites. The erysipelatous rash may also be mistaken for true erysipelas, but the long febrile period of erysipelas will soon serve to distinguish it.

Treatment. During the attack, rest with elevation of the affected part, cold compresses, or hot fomentations, may be used; ¼-grain (0.01 Gm.) doses of calomel at hourly intervals, followed by large doses of magnesium sulphate as soon as the calomel acts, should invariably precede other medication. Opium in suitable form may be given to relieve the pain.

ELEPHANTIASIS.

Elephantiasis is a hypertrophy of the tissues under the skin in areas of the body affected by lymph-stasis, resulting in enormous enlargement of the legs, arms, or other parts of the body, and is by far the most striking, as well as probably the most frequent, manifestation of filarial infection.

Of the numerous synonyms for this disease the following are in vogue: Cochin leg, Barbados leg, elephantiasis arabum, elephant leg, hypersarcosis, glandular disease, and Phlegmasia Malabarica.

Elephantiasis is found in the legs, the scrotum, the vulva, the arms, the breast, and rarely in other regions. It occurs wherever filarial disease is present, and it is usually not found in areas in which filariæ are absent. At times cases of elephantiasis have been reported in filaria-free areas, but they were only isolated cases, and the infection was probably



Fig. 16.—Elephantiasis of the lower extremities. Front view. (Author's case.)

contracted in endemic areas. Bahr[144] found that in the Fiji Islands 3.56 per cent. of the population examined were afflicted with elephantiasis, and that 52.5 per cent. of the entire population showed evidence of filarial disease in some form. It prevails extensively in nearly all the islands of the South Pacific, the West Indies, the tropical Americas, tropical Africa, and tropical Asia. In Samoa it is a veritable scourge, and probably affects a larger percentage of the population than in the Fiji Islands, although there are no reliable data available as to the exact extent of its prevalence.

Manson[145] states that in "95 per cent. of the cases the lower extremities—either one or both—alone, or in combination with the scrotum or arms, are the seat of the disease. The foot and ankle only, or the foot and leg, or the foot, leg

and thigh, may each or all, be involved." The arms are seldom involved, and still more rarely the mamma, vulva, and circumscribed portions of the limbs, trunk or neck.

The disease, regardless of the area which is affected, commences with lymphangitis, and soon results in dermatitis and in inflammation of the deeper cellular tissue. The symptoms of an acute attack are the same as those described under filarial lymphangitis, including the erysipelatous rash. These processes are always accompanied by fever. The fever may last for from several days to several weeks. After subsidence



Fig. 17.—Same patient as Fig. 16. Side view. (Author's case.)

of the acute symptoms the skin and subcutaneous fascia of the affected area do not quite return to their normal proportions. Some exudate remains unabsorbed, and a certain amount of permanent thickening remains. Recurrences of the foregoing process may take place at intervals of several weeks or a month, or even six months or more may elapse before another acute attack occurs. Each time a little more bulk is added to the affected areas, and the part in consequence steadily increases in size. In this way there is gradually built up an enormous leg, or arm, or other anatomic unit. In a few cases there are no further acute attacks, and the disease remains stationary. After one or two attacks the skin over the affected area becomes rough and coarse. The papilla and glands may either be hypertrophied or atrophied. The hair becomes coarse, and the nails rough, thick and

deformed, no doubt because of nutritional disturbances. When extensive swelling occurs over the joints there is very often considerable interference with movement. The swelling, as a rule, is hard and dense, and only pits slightly on pressure.

The dorsum of the foot commonly becomes swollen and edematous. The *débris* of the desquamating epithelium, with the excretions of the skin, are liable to accumulate, and to give rise to foul-smelling discharges, and at times ulcers may form. Some cases of elephantiasis may occur without there having been any attack of fever observed. The muscles, nerves or bones are not necessarily diseased, although at times degenerative changes or atrophy may occur from pressure.

Elephantiasis of the Legs. As a rule, elephantiasis of the lower extremity is usually confined to the section below the knee. However, elephantiasis of the thigh is sufficiently common. The size of the leg is sometimes enormous, and fully as large as that of an elephant's leg, and it is this symptom that gave the disease its name. There is always great disturbance of the dermal appendages. The skin becomes rough, the hairs coarse, the nails thickened and deformed. Ulcers are frequently encountered, and may be located in any area. They often follow slight injuries, and, as a rule, are persistent and do not respond readily to treatment. Briefly, the symptoms, with few exceptions, are the same as those described under general elephantiasis.

Treatment. A satisfactory method of treatment has not yet been evolved. In India operative treatment, especially the removal of large masses of tissue, has been very generally done. Fibrolysin injections have been used by Castellani, and at the Hamburg Institute for Tropical Diseases, with a fair amount of success. The patient is placed at rest in bed, and 2- to 4- mil (32.4 to 64.8 *m.*) injections of fibrolysin (thiosinamin and salicylic acid) are given daily for periods varying from three to six months. Occasional intervals of a few days are allowed between treatments. The injections are given with a hypodermic syringe, deeply into the affected parts or into the gluteal region. After the injection the limbs are tightly bandaged. At Hamburg it has been customary to

apply a rubber bandage over the cotton bandage, in order to bring about even pressure. On account of the extreme heat in the tropics rubber bandaging is, as a rule, very uncomfortable. It is especially recommended by Castellani in the verrucose type of the disease. Massaging of the limb in the elevated position appears to be efficacious. Surgically, numerous operations have been devised. Most of these are based on the excision of wedge-shaped strips of tissue of varying lengths. As a rule, surgical operation must be preceded by medical treatments on the lines given above, and no operation attempted during acute febrile periods. Southey's tubes, which consist of a number of small cannulas, are inserted under the skin, in order to permit the free discharge of fluid. Great reductions in the size of the limb can frequently be obtained by this method. It is doubtful, however, whether any permanent decrease in the size of the limbs can be brought about. When the limbs become of sufficient size to prevent a patient from getting about comfortably, amputation may be indicated. Some patients are much better off with an artificial leg and foot than with the enormous deformity and weight of elephantiasis.

Elephantiasis of the Scrotum. The scrotum may attain enormous size. A weight of 20 pounds (9.07 Kg.) is customary, and of 50 pounds (22.67 Kg.) is not infrequent. Manson states[146] that the largest reported weight is 224 pounds (101.6 Kg.). The onset of the disease and its general progress is the same as in other parts of the body.

Treatment. The complete excision of all the affected tissue of the scrotum probably produces more satisfactory results than the treatment of elephantiasis in any other part of the body. The greatest care is necessary to perform the operation under the most aseptic conditions. Infections are very likely to occur, and special attention is required properly to cleanse the diseased scrotal areas. Various operations have been described, for the details of which a textbook on surgery should be consulted.

Elephantiasis of the Vulva. This condition is essentially the same as that of elephantiasis of the scrotum. The hypertrophy may implicate the labia majora or the clitoris. This condition is rare. According to Manson,[147] vulval tumors

may weigh from 8 to 10 pounds (3.62 to 4.53 Kg.), or even more.

The treatment consists in the surgical removal of the tumor.

Elephantiasis of the Breast, Arm, Scalp and Other Regions. Elephantiasis of the breast is very rare. That of the arm is even rarer, and that of the scalp is seldom seen. Other regions of the body may be affected, but only in the very rarest instances.

The treatment is the same as that indicated for elephantiasis of other regions.



Fig. 18.—Filaria nocturna. (*Da Costa.*)

Filarial Organisms. As a rule, the filariæ can be found in the blood only during the night hours. However, there are exceptions to this rule. Bahr,[148] for instance, working in the Fiji Islands, found the microfilariæ as readily during the daytime as at night. In a fresh-blood specimen the *Microfilaria bancrofti* appears as minute, transparent, colorless, snake-like micro-organisms, which do not change their position on the slide, although they wriggle about in great activity, constantly agitating and displacing the cellular elements of the blood (see illustration). In the course of a few hours the movement ceases. The microfilariæ are long, slender and cylindric. One extremity is abruptly rounded, and the other gradually tapers to a fine point. The filaria is about 0.3 millimeters (0.01 in.) in length, and 0.008 to 0.001 (0.0003 to 0.00004 in.) in diameter; in other words, approximately the diameter of

a normal erythrocyte. As a rule, they begin to appear in the peripheral circulation during the early evening hours and increase in numbers until midnight, after which they gradually decrease. From 300 to 600 may be found in a single drop of blood. This periodicity may be maintained over a period of years. If the subject sleeps during the day and is awake during the night hours, the periodicity is reversed. Manson states that during their temporary absence from the peripheral circulation, the microfilariæ could be found during the daytime in large numbers in the larger arteries and in the lungs. Man is the definitive host; the mosquito the intermediary host.

The adult worm is a long, hair-like, transparent nematode from 7 to 10 centimeters (2.7 to 3.1 in.) in length. The male and female usually lie closely together, and are often intertwined. The female filaria is the larger, both in length and diameter. In both sexes the oral end is slightly tapering. The tail also tapers to comparatively small dimensions, but it is bluntly rounded off. The male worm can be distinguished by its smaller dimensions and its disposition to curl, and also by the tendril-like tail. In some sections, especially in Africa, large numbers of *Filaria perstans* may be found. Sometimes they are found in association with the *Filaria loa* and the *bancrofti*. Manson[149] also describes *Filaria demarquayi* and *Filaria ozzardi*.

Death seldom takes place primarily from filarial disease. At times, however, through rupture or perforation of important organs, as, for instance, the peritoneum, secondary conditions like peritonitis or other infections may occur which result in death. With the exception of elephantiasis of the scrotum, vulva and other regions in which a complete excision can be made, the prospects for a cure are not encouraging, and seldom take place. In a fair percentage of cases the disease soon becomes arrested. The only difficulty remaining is the resulting deformity.

To avoid mosquito-bites is the most important consideration in the prophylaxis. The consumption of food, like bananas, for instance, upon which infected mosquitoes have fed, may be a possible, although not a probable, source of infection. As the mosquitoes, which are mostly concerned

in the transmission of the disease, are more or less domestic in nature and breed in artificial containers and drainage ditches, antimosquito measures, even of a modest character, may do much to free a community of the type of mosquitoes responsible for the conveyance of filarial disease. Minor drainage operations and the oiling of stagnant water should always be done in the vicinity of habitations in regions in which filarial infection is endemic. Persons residing in filarial countries should invariably sleep under mosquito-nets. This is an important protective measure, on account of the fact that the filaria-carrying mosquito usually flies only at night.

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The Intoxications

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FOREWORD.

UNDER the heading of The Intoxications the writer has included the management of the various morbid conditions consequent to the introduction into the human body of certain chemical agents possessed of a poisonous action. Of these mercury, arsenic, lead, phosphorus, silver, copper, zinc, and tin are prominent examples, and numerous fatalities result from their influence, both when taken with suicidal intent and in connection with industrial pursuits.

The practising physician also is not infrequently called upon to treat poisoning by illuminating gas, so that this condition is described in some detail. The same is true of poisoning by opium, alcohol, and cocain, and here, in addition, the medical adviser is concerned, not only with the active emergency, but with the after-treatment and the important social problems that invariably demand adjustment.

Food poisoning is approached from the dual viewpoint of prevention and care of the active toxemia, and this plan is followed in the consideration of thermic fever and heat exhaustion.

Aside from describing the principles of treatment in vogue at the present time, the leading features of diagnosis and symptomatology of the various toxic states are briefly dealt with, along with other details of the clinical picture relating mainly to topics such as personal predisposition, the physical state of the subject, and the disturbances of the several body functions. A consistent attempt has been made to furnish the reader with a helpful working guide to the treatment of the commoner intoxications, and to this end theoretic discussion has been minimized and practical therapeutic measures have been emphasized, in so far as the nature of the condition in question warranted this plan of procedure.

GENERAL CONSIDERATIONS.

Toxicology is the scientific study of poisons, their sources, properties, actions, their detection and the treatment thereof.

Authorities differ as to the substances that should be included under the term poison.

Vaughan defines a poison as a substance of definite chemical composition which, by virtue of its constitution is capable, when brought in contact with the tissues of the body, of modifying the cellular activity of one or more organs in such a way as to impair health or destroy life.

Actions of Poisons. The modes of actions of poisons may be classed as local and constitutional. To the former class belong those poisons which only impair or destroy the tissues with which they come directly in contact; and to the latter group are referred poisons in which the symptoms are due to its presence in the blood and its effect upon the various organs of the body.

Local and constitutional poisons affect the tissues with which they are in contact, and have further action due to absorption, *e.g.,* arsenic and carbolic acid. The ordinary symptoms of poisoning are due to absorption, but in order that any substance should give rise to these it is necessary that it should be taken into the blood and carried throughout the circulation.

Conditions Influencing the Action of Poisons. The size of the dose taken, the method of administration, the physical state of the poison, age, habits, previous health and idiosyncrasy are factors which determine the wide difference in the symptoms induced by poisons in different individuals.

The Size of the Dose. As a rule, the larger the dose of the poison taken, the severer are the symptoms, and the more likely to prove fatal.

Sometimes a large dose induces vomiting or purgation, by which the poison is promptly expelled from the body, when a smaller dose, not sufficient to produce emesis, results in a marked toxemia.

If a large quantity of arsenic is quickly absorbed, the usually prominent gastro-intestinal symptoms may be wholly wanting, while the effect upon the central nervous system predominates.

When ½ ounce (15 mils) of spirits of turpentine is taken it may cause symptoms referable to the gastro-intestinal tract so

severe that the vomiting and diarrhea may rid the body promptly of the turpentine; on the other hand, 1 dram (3.75 mils) of spirits of turpentine will not cause sufficient gastro-intestinal irritation to cause it to be promptly eliminated, but the slow action upon the kidneys may result in permanent injury thereto.

The Method of Administration. Poisons can be introduced into the body in a number of ways. Any means may be employed by which it reaches the general circulation, viz., by introduction into the stomach, or rectum, and from absorption through the lymphatics, or subcutaneous injections.

Rapidity of action depends on the mode of administration. If a poison enters the body slowly the kidneys and other excretory organs very often can dispose of it before serious effects are produced. Rapidity of action largely determines the effect of the poisons on the tissues; nevertheless the avenue by which the poison is introduced is likewise an important factor.

Substances. Arsenic, when injected subcutaneously, exerts a marked local effect with but slight constitutional symptoms; on the other hand, the same amount of arsenic, given or taken by mouth, produces graver results.

The Physical State of the Poison. Upon the physical state of the poison depends largely the question of absorption.

Gases are more rapidly absorbed than liquids, which are in turn more rapidly absorbed than solids.

The fineness of the powder is a determining factor in the variation of the effects of poisons.

Thus, the finer the powder the more rapidly it passes into solution, which is essential if the poison is to be absorbed. Likewise the finer the powder the more surface of poison is exposed whereby it can exert its deleterious local action. The degree of concentration influences the effect of poisons.

A mineral acid in concentrated form may destroy all tissues with which it comes in contact, while the same amount of acid freely diluted with water may be relatively harmless.

Physical Condition of Recipient. As a general rule, children are more susceptible to poisons than adults. Children stand relatively larger amounts of arsenic and chloral, and, on the other hand, bear opium badly. The aged are more readily poisoned than those in middle life.

The previous poor state of the individuals' health renders them more liable to the effects of poison. The exceptions to this general statement are those suffering from colic, peritonitis, and delirium tremens, who display a marked tolerance for opiates.

A person who is continually taking sublethal doses of any poisonous substance may, in some cases, acquire a degree of tolerance, and larger amounts are necessary to obtain the effect of the drug, *e.g.*, opium. Other substances in which a tolerance may be acquired are cocain, tobacco and chloral. Personal idiosyncracy may account for the untoward effects of full therapeutic doses of quinin, mercury, bromid, and less frequently, a number of other drugs.

Classification of Poisons. Any classification attempted must, of necessity, be more or less arbitrary, especially if based upon the physical action of the poison.

Poisons have been classified according to their origin and chemical composition, *i.e.*, inorganic and organic; and animal, mineral and vegetable.

Taylor classes all poisons under two heads:

Irritant.
Neurotic.

Irritants. All poisons which have an irritating and destructive action upon the tissues with which they come in contact, and, when taken by way of the alimentary tract, are followed by greater or lesser amount of irritation and destruction of the tissue of that canal are classed as irritants.

As a result locally the ingestion of poisons is followed by nausea, vomiting and diarrhea accompanied by blood.

Irritants are further subdivided into irritants proper, and include all chemicals whose poisonous effects largely, if not entirely, depend upon their irritant and destructive action on tissues with which they come in contact. Stronger acids and alkalies belong to this group.

The second class of irritants are the specific irritants whose poisonous action depends not only upon their irritating effect, but on specific physiological results which follow the taking of the poison. To this class belong such metals as arsenic, antimony, mercury, copper and lead; non-metallic poisons, as chlorin, bromin, iodin, and phosphorus; vegetable irritants such as

oxalic acid, tartaric acid, croton oil, elaterium; animal poisons, as cantharides and albumins developed during putrefaction.

Neurotics. Under the general heading of Neurotic Poisons are included a group of poisons which act specially upon the great nerve centers, the symptoms presented being those dependent upon their action upon the brain and spinal cord. The symptoms of this group are drowsiness, headache, delirium, coma, convulsions or paralysis.

Subdivisions of neurotic poisons: cerebral neurotic, including anesthetics and depressants, spinal neurotic or tetanics, and cerebrospinal neurotics or deliriants.

Symptomatology of Poisons. A wide range of symptoms are to be looked for as the result of poisoning, and upon their prompt recognition and association depends the success of the treatment.

Among the symptoms more or less common to poisons are the following:

Vomiting and Purging. These symptoms suggest that nature is endeavoring to rid the gastro-intestinal tract of irritating substances, and are, indeed, common in practically all forms of poisoning. The prompt appearance of these symptoms following the onset of poisoning is often life-saving. The vomiting and purging usually are present at the same time, although vomiting may occur independently of purging, for example, in opium poisoning. The intensity of one or both of these symptoms varies greatly.

The Temperature. No great change occurs in the temperature, but it is not uncommon to find the temperature subnormal, seldom increased except in some conditions characterized by tetanoid convulsions, *e.g.*, strychnin poisoning. The shock to the system as the result of vomiting, or pain produced by passing a stomach-tube, has caused the temperature to vary. The increase in temperature is seldom more than several degrees, while in severe or fatal cases the temperature may drop to 95° F. (35° C.).

The Pulse. As a general rule the rate of the pulse in cases of acute poisoning is increased. This condition is dependent upon the amount of shock present, and the depressant action the poison may have on the heart directly or through the nerve mechanism controlling the heart. Poisons that act directly upon the respiratory center may prove fatal without a

change in the rate of pulse, which continues to beat some time after the respirations have ceased.

Respiration. Dyspnea is the most common symptom recognized in cases of acute poisonings, which may be due to mechanical obstruction, as in edema of the glottis from the local action of a corrosive poison. Paralysis or spasm of the respiratory muscles, the former observed in chronic lead poisoning, and the latter in strychnin poisoning, accounts for dyspnea and variation in the respiratory rate.

Cerebral Symptoms. These symptoms, common in many other diseases, are likewise prominent in many of the cases of acute poisonings. It is, therefore, important to seek the cause of this group of symptoms.

Illusions, hallucinations and delusions may follow poisonous doses of a number of drugs—opium, chloral, quinin and salicylic acid. Convulsions have been noted in belladonna and strychnin poisoning. In the latter stages of many of the poisons other than narcotics, stupor and coma supervene.

Vasomotor Disturbances. Apparently the vasomotor center is easily affected by poisons, as the result of which changes in the heart-action and respiration occur. Variations noted in the skin are alternate or continuous flushing and blanching, and frequently the skin is cold or clammy, or dry and red.

Motor Disturbances. The muscular spasm and convulsions in tetanus and strychnin poisons are very prominent symptoms, and of diagnostic import.

Groups of muscles may be paralyzed, such as the musculospiral palsy associated with plumbism.

Skin Lesions. Many drugs produce, if taken over a great length of time, or if a hypersensitiveness is present, a variety of more or less uniform skin lesions, such as the papular and acniform rashes when iodids and bromids are taken; the characteristic rashes following the introduction into the body of belladonna and iodoform in poisonous doses. As the result of prolonged use of silver, a peculiar bluish color of the skin is noted, due to the subcutaneous deposit of silver called argyria.

Sensation Abnormalities. The neuritis of chronic arsenical poisoning is sufficiently distinct to be considered a factor in cases of neuritis presenting themselves for diagnosis and treatment.

The variation in sensation associated with chronic cocain poisoning is of diagnostic aid.

The Eye. The examination of the eyes is diagnostic in cases of poisoning, frequently giving important clues in identifying the poison. The pupil in opium poisoning is pin-point, and, just prior to death, dilates.

Poisonous doses of santonin produce yellow vision; and ingestion of wood alcohol in sufficient quantity produces blindness.

Atropin produces dilatation of the pupil, taken internally as such, or a belladonna preparation, like a solution of atropin, dropped into the eye will produce a similar effect.

Ear. Quinin in very full doses produces ringing noises in the ear; and the buzzing caused by salicylates or salicylic acid is very characteristic.

The Diagnosis of Poisoning. The detection of a case of poisoning is not always a simple task, especially if given or taken with criminal intent. Accidental poisonings are usually promptly reported to physicians, with the diagnosis very clear.

In every case of suspicious poisoning, any food, drink or medicine, vomitus and excreta, should be inspected, and, if necessary, samples should be taken for chemical or microscopical examination.

Vomitus. The vomitus may show something of value by its appearance, odor and color, such as the odor of laudanum, carbolic acid, or hydrocyanic acid, which is characteristic. Portions of the undissolved poison may be present in the vomitus, as particles of bichlorid of mercury tablets. The vomitus in phosphorus poisoning is luminous in the dark.

The odor of the exhaled air may promptly suggest the variety and presence of a poison.

The urine should be obtained, and promptly submitted to examination in every case of poisoning.

Sulphonal and trional in poisonous doses, or given over a great length of time, make the color of the urine a rich Burgundy red. The urine alkalinized after santonin poison becomes bright red.

Iodoform or iodin can be detected in the urine by chemical tests.

Phenol and allied bodies render the urine a dark green or smoky color, spoken of as smoky urine.

Blood, albumin and casts appear in the urine as a result of irritant poisons, as cantharides, chlorate of potash, turpentine, or any substance that causes an acute nephritis. The urine may have the odor of violets after turpentine poisoning.

It is likewise important to bear in mind that not every person found unconscious in a bedroom, or bathroom, with bottles of poison about, is necessarily suffering from attempted suicide by poisoning. Uremia or diabetic coma has thus been mistaken for cases of poisoning. An examination of the mouth, hair, face, lips, and tongue may often disclose marks of corrosive poisoning.

In cases of poisoning with suicidal intention, the patient's clothes and the immediate vicinity about the patient should be searched for clues to identify the poison.

The examination of the skin of chronic morphin and cocain habitues usually reveals the prick-points of hypodermic needles. When poison is unsuspectedly taken with food or water, the evidence of its occurrence may be furnished by the fact that a number of persons are suddenly seized at about the same time under similar conditions. The evidence upon which a case of poisoning is based will be, first, the symptoms in a patient while alive; second, the symptoms discovered at autopsy; third, the result of chemical analysis; and fourth, moral or circumstantial evidence.

PRINCIPLES OF TREATMENT OF POISONING.

1. Removing poison from the body.

2. Rendering the poison inert by forming a new chemical compound.

3. Neutralizing the effect of a poison by the administration of a drug having the opposite action.

4. Treatment of special symptoms.

Removing the Poison from the Body. This may be accomplished by the use of the stomach-tube; by giving purgatives or emetics.

The stomach-pump is, in the majority of instances, the most effective means we have of removing poison. The use of the tube may be of value even if the drug is given or taken

hypodermically, as in hypodermic morphin poisoning the drug is eliminated from time to time into the stomach.

Following the ingestion of corrosive poisons, because of spasm or great pain, or danger of perforation, the use of the stomach-tube is contraindicated.

Simple warm water may be used, but the addition of permanganate of potash, or tannic acid in the cases of alkaloidal poisons, the white of egg in mercury poisoning, and ferric hydroxid in arsenic poisoning, greatly aid in antagonizing the poisonous effects of the drug.

Emesis. This is frequently the first remedial measure applied, and very effective, although not to be preferred to the use of the stomach-tube provided that facilities for both are at hand.

Domestic remedies, and means known to every one, are usually available, and can be used until a stomach-tube is obtainable. In the case of large particles of poisonous substances remaining in the stomach, the caliber of the stomach-tube may not permit the aspiration of the poison, and therefore it may be necessary to use emesis.

Tickling the fauces or the post-pharyngeal wall usually produces emesis. Emetics are contraindicated in corrosive poisons. Emetics are often useless in poisoning by narcotics, or by substances which diminish the sensibilities of the mucous membrane of the stomach.

Emetics act locally upon the stomach, or centrally upon the vomiting centers of the brain.

The following are emetics commonly used that act locally:

Mustard and Water. A tablespoonful (14.1 Gms.) of mustard in a glass of warm water.

Salt and Water. One or 2 tablespoonfuls (15 or 30 Gms.) may be given in a tumbler of warm water.

Zinc Sulphate. Twenty or 30 grains (1.3 or 2 Gms.) dissolved in 4 ounces (120 mils) of warm water may be given, and repeated if necessary.

Ipecac. In the form of syrup, 1 or 2 ounces (30 or 60 mils) repeated if necessary in fifteen minutes, for an adult, or 4 to 6 drams (15 to 24 mils) of the wine.

Emetics acting centrally are of use in cases of narcotic-poisoning. The principal objection is the great amount of depression produced by them.

Apomorphin, best administered hypodermically, in doses $\frac{1}{10}$ grain (0.006 Gm.). Tartar emetic should not be used because of great depression produced.

Purgations. A brisk purge is an appropriate means of aiding in the elimination, and often the saline may be put in the stomach through stomach-tube while *in situ.* A purge is particularly indicated when material has passed from the stomach into the intestines; an enema given at the time an inclination of the bowels to move is experienced is very advantageous.

Poisons may be rendered inert by forming therefrom an insoluble compound, as in the case of sulphates, given in lead poisoning. Alkali poisons are neutralized by acids and *vice versâ.*

Solutions of permanganate of potash oxidize, and thereby render less poisonous the effects of alkaloids. The albuminates of the metals are rather insoluble, but should not be allowed to remain in the stomach or intestines, as they are absorbed slowly, whereupon symptoms of poisoning reappear.

Neutralization of the effects of a poison may in a sense be effected by administering a drug having the opposite action.

No one drug has exactly the opposite action of another, but there are drugs whose principal action is exactly the opposite of that of another. The action of drugs, while opposite in effect, may not be manifest simultaneously, one or the other acting slowly.

Because a drug is theoretically antagonistic does not mean that it is the best antidote; and the fact that it is the best antidote is not proof that the reverse is true.

A few examples of the use of drugs having antagonistic actions are noted.

Atropin and physostigmin. Atropin in small doses is an excellent antidote to poisoning by physostigmin.

Atropin and pilocarpin. Pilocarpin forms a sound antidote to atropin.

Morphin and atropin. Atropin acts as an antidote by neutralizing the depressing action of morphin upon the respiratory center and higher cerebral centers.

Strychnin and chloral. Chloral, which acts to a certain extent upon the spinal cord, but chiefly on cerebrum, is a valuable antidote to strychnin.

Other instances of antagonism in drug action are aconite and digitalis, and hydrocyanic acid and chloroform.

Treatment of Special Symptoms. This subject will be treated under the discussion of each poison.

MERCURIAL POISONING.

Poisoning by mercury manifests itself as the acute and the chronic form. The vapor of metallic mercury is highly toxic, and chronic poisoning is a frequent occurrence among workers engaged in smelting ores contaminated with mercury.

Mercuric chlorid owes its corrosive action to its affinity for the proteins. The mercury is absorbed into the blood-stream as the albuminate if absorbed by skin or mucous membrane, regardless of the kind of salt of mercury taken or given.

Gastro-intestinal irritation and inflammation are encountered as the dominant pathologic lesion in acute mercurial poisoning. Various degrees of inflammation may be present, and hemorrhage may be associated with erosions of the mucous membrane, or even perforation of the stomach or intestines. The corrosive action of the bichlorid of mercury produces the effects that are noted when acids or alkalies are swallowed.

If the poisonous dose of mercury has caused death in a few minutes, histologic changes are not prominent. Discoloration and ulcers of the gums have been noted, and at autopsy fatty degeneration of the viscera is seen, especially in the kidneys and in the liver. Often deposits of lime are found in the kidneys, due to the fact that in mercurial poisoning calcification occurs in the renal epithelium. The researches of E. Ludurg have shown that the places of predilection for the deposit of mercury are the kidneys, liver, and the walls of large intestines, in the order named. Uremia and the lesions of neuritis have been noted.

The large majority of cases of mercurial poisoning are caused by bichlorid of mercury. The symptoms complained of are a burning sensation in the mouth and throat, accompanied by constriction due to the local action of the poison.

When the poison reaches the stomach, pain is complained of, and usually severe vomiting follows. Often the vomitus is

streaked with blood. The vomiting is followed by purging, and hemorrhages from the bowels may occur.

Accompanying these symptoms usually are the evidences of collapse; the pulse becomes rapid and feeble; a cold, clammy skin; thirst is complained of; respiration is rapid and labored; and muscular cramps are not uncommon. Death, preceded by coma or convulsions, may end the scene.

The symptoms of mercurial poisoning vary widely. An individual may take a poisonous dose and not be treated promptly, experience slight distress upon taking the poison by mouth and swallowing it, and not until a week or ten days later pass into coma and die, with or without convulsions; on the other hand, through violent vomiting or purging, the system may eliminate the poison, and no ill effects be permanently experienced.

Slight chronic mercurial poisoning, which is not very common, due to too long continued treatment or too large doses of mercury, is manifested by salivation, sore gums, foul breath, abdominal pains, and not uncommonly diarrhea. Necrosis of the bones of the jaws has been observed. Skin eruptions may be noted; a slight fever and headache.

TREATMENT.

In an acute case of mercurial poisoning, large quantities of an albuminous substance, such as the white of egg, should be given by mouth. In this manner the mercury unites with the albumin and forms the albuminate of mercury, which is not an inert substance, but which should be removed from the stomach by means of the stomach-tube.

The value of an albuminous substance is that it lessens the corrosive action of the mercury, and hence protects the mucous membrane of the stomach. Albuminate of mercury is highly poisonous but possesses no corrosive action.

With the advance in the knowledge and results of treatment, aided by the studies of Lewis and Rivers, Lambert and Patterson, Bellfield and others, the condition does not appear so hopeless as was prognosticated in the past.

A great deal of new information has thrown light upon the functional activities of the kidneys, as recorded by the phenolphthalein test; estimation of the urea and non-protein nitro-

gen of the blood, which in turn applied to the study of the kidneys in mercurial poisoning, furnishes a rational background upon which to base the character and effectiveness of treatment.

Briefly, elimination is the factor essential to secure favorable results, and every effort is directed to this end in the treatment of mercurial poisoning.

Since the excretion of mercury into the stomach and intestines, as well as elimination by the sweat-glands, has been definitely established, it is important that the stomach and intestines be washed out frequently. Frequent sweats induced by hot packs are most beneficial.

MacNider has shown that the administration of alkalies is capable of partly protecting the kidneys from the effects of the salts of heavy metals like uranium, which leads to pronounced nephritic disease, as mercury does.

The results of the estimation of the non-protein nitrogen of the blood indicate the retention of nitrogenous waste.

Lewis and Rivers therefore urge the administration of carbohydrate in the form of glucose. The glucose is supposed to act in the capacity of a protein-sparing agent.

Mercurial stomatitis is best treated with a mouth-wash consisting of chlorate of potash, 7½ grains (0.5 Gm.) to 1 ounce (30 mils) of water.

The teeth should be kept clean by the use of an alkaline tooth-powder or wash used on a toothbrush with soft bristles.

Belladonna preparation given internally will lessen salivation, if very annoying.

Astringent mouth-washes composed of alum and tannic acid are useful.

Fantus recommends the following antidotal treatment: immediate administration of a tablet composed of sodium phosphate 0.36 Gm. (6 gr.) and sodium acetate 0.24 Gm. (4 gr.). If this is not available, give the following: sodium hypophosphite 100 Gms. (3 oz. 230 gr.), water 10 mils (160 *m.*), and hydrogen peroxid 5 mils (80 *m.*). If the amount of the poison taken be known, ten times as much of hypophosphite should be given as poison was taken. As this might require a large and possibly harmful amount of hypophosphite, it should immediately be followed by a copious lavage with a very dilute

solution of the antidote. This may be followed by a safe dose of the antidote, which is to be retained, and which can be repeated every eight hours for several days. This antidotal treatment can be combined with some eliminant treatment as suggested by Lambert and Patterson.

Elimination is vigorously sought after, and to its attainment is attributed the success of this mode of treatment.

The stomach and the colon are washed out twice daily with warm normal saline solution. The patient is placed on a liquid diet consisting of 8 ounces (250 mils) of milk every two hours to be alternated every two hours with 8 ounces (250 mils) of the following mixture:

Potassium bitartrate,	
Sugar, of each	ʒj (3.9 Gms.).
Lactose	ʒiv (15.6 Gms.).
Lemon juice	fʒj (30 mils).
Boiled water	fʒxvj (500 mils).

In addition to the large amount of water given by mouth a solution of 1 ounce of potassium bitartrate (31.2 Gms.) to a pint (500 mils) of water is given continuously by rectum.

Daily hot packs are given to the patient. This treatment should be continued and its efficiency measured by the phenolsulphonephthalein test until no mercury can be detected in the excretion fluids.

ARSENIC POISONING.

Arsenic poisoning resembles conditions noted in other diseases. A positive diagnosis of arsenical poisoning or its exclusion cannot be made upon symptoms alone, and the presence of arsenic by chemical examination must be determined before positive statements can be made.

Acute Poisoning. After a poisonous dose of arsenic has been taken into the body, symptoms usually referable to the gastro-intestinal tract generally appear within two hours. A sensation of faintness in the epigastrium is not uncommonly an early symptom. This is followed by retching, nausea and vomiting, which are present throughout the attack or until death intervenes. The vomiting is accompanied by a sensation of burning and dryness in the mouth, throat and stomach, and a marked thirst.

After the contents of the large bowel have been expelled the stools become more and more choleraic in character, and are designated as "rice-water."

Extreme prostration as the result of continuous vomiting and purging ends in collapse, with pallor, cold, clammy skin, sunken eyes, rapid-running pulse, and shallow respiration.

Unless promptly treated, the patient usually dies within twenty-four hours, death being preceded by coma and convulsions.

Occasionally, when a poisonous dose of arsenic has been rapidly absorbed, death may occur promptly, after a state of collapse, with no gastro-intestinal symptoms. Such a condition has been known to occur when a soluble salt of arsenic has been introduced into an empty stomach.

The mortality in acute arsenical poisoning varies, depending on the promptness and efficiency of treatment, and is about 50 per cent. Death may occur in less than one hour after the poison has been taken, and the patient seldom lives twenty-four hours in fatal cases.

Subacute Poisoning. If smaller quantities of the poison are introduced into the body, or if the effect of a large dose of arsenic has been mitigated by treatment, the symptoms characteristic of acute arsenical poisoning come on more slowly, are milder in character, and continue for a greater length of time.

The gastro-intestinal symptoms are prominent, but to a lesser degree than in the acute form of poisoning. If death occurs, it usually takes place within from two to ten days after the poison is ingested. The skin becomes dry and warm, and a rash may develop. The vomiting may be intermittent in character. At times a temporary remission of the symptoms occurs about the third day, and this is followed, as in a case of phosphorus poisoning, by jaundice and delirium.

Disturbances of motion are usually confined to the extremities. Muscular atrophy accompanies the paralysis of muscles. Disturbances of sensation accompany those of motion, and may predominate. Pain is usually present, and may be distressing in character.

A neuritis similar in character to that caused by toxic agents is present in a number of cases of chronic arsenical poisoning.

Chronic arsenical poisoning may result from the long-continued administration of arsenical preparations, and is manifested by disturbances of the gastro-intestinal tract and puffiness under the eyes. A slight fever, mild delirium, dry tongue, rapid pulse, and often a cutaneous eruption, usually predicate death.

Chronic Poisoning. Frequently the skin bears the brunt of chronic arsenical poisoning. Arsenic rashes may be of the erythematous, papular, vesicular, pustular, ulcerative and gangrenous types. Pigmentation of the skin has also followed prolonged use of arsenic.

Poisonous doses of arsenic have a very destructive effect on the coloring matter of the blood. In some cases transitory or permanent paralysis of the muscles, or some weakening thereof has been noted.

In addition to the foregoing symptoms, prolonged arsenical medication may result in other lesions of the skin indicative of cellular proliferation, such as hyperkeratosis of the hands and feet.

In the acute form, inflammation of the gastro-intestinal tract, nephritis and fatty changes in the muscles and the viscera, especially the liver, constitute the principal lesions; in the chronic form, pigmentation of the skin and lesions of the nervous system, notably of the peripheral nerves, are common.

The lesions induced by the arsenic in the liver and kidneys are easily recognized by microscope; only exceptionally have fatty changes advanced sufficiently to be recognized by the eye.

In acute arsenical poisoning hemorrhagic areas may be found in the alimentary tract, muscles, pancreas, lungs and serous membranes.

TREATMENT.

In acute poisoning the stomach should be emptied by the stomach-tube, even though spontaneous emesis has taken place. Various arsenical preparations in powder form closely adhere to the stomach wall, hence the necessity of thoroughly washing out the stomach with an abundance of water. The best chemical antidote is the freshly prepared ferric hydroxid, or the mixture of ferric hydrate with oxid of magnesia, official in U. S. Pharmacopeia.

It is a very good practice to keep the iron solution and the magnesia mixture in separate bottles in a physician's office, so

that they can be immediately mixed if the emergency presents itself.

The iron and magnesia preparation can be given freely, a tablespoonful (15 mils) repeated every three or four minutes for one-half hour. These substances act by forming insoluble arsenites; as in other irritant poisons, diluents, demulcents, and opiates are usually indicated.

LEAD POISONING.

Poisoning by lead, or plumbism, is by far, next to alcohol, the most important intoxication from a clinical standpoint. Acute lead poisoning, either accidental or with criminal intent, is extremely rare, in comparison with the chronic form encountered in workers in the lead industries.

Considerable improvement in the working conditions of employees in lead occupations has resulted in fewer cases of lead-intoxication at present than ten years ago. There is need, however, for further lessening the dangers of these occupations.

The symptoms of plumbism, in most instances, are more or less common, but not constantly present. Acute lead poisoning is usually accidental, as the result of taking the acetate of lead, which may be followed by serious results, which eventuates at times in death, usually within two or three days.

The symptoms are those of gastro-intestinal origin, and consist of vomiting, retching and colicky pains, with obstinate constipation. In some cases the vomiting is protracted, and is of a bloody character. The pulse becomes rapid, irregular; the breathing quickens, becomes shallow, and coma may precede death.

If the moderate intoxication of lead is continued over a greater or lesser length of time, graver symptoms and conditions may develop, such as convulsions, severe headaches, cardiovascular symptoms, nephritis, arteriosclerosis, palsies, and even paresis. Usually the symptoms are preceded by some intestinal colic.

The subject of chronic lead poisoning may be complaining for a number of years of poor health of an indefinite nature. Frequently death resulting from nephritis is superinduced by lead.

Abdominal colic, when occurring in painters and others engaged in the "lead trades," should always suggest a chemical

examination of the urine for lead, unless the diagnosis otherwise is very evident.

A grayish or black deposit of lead sulphid near or at the free margins of the gums, spoken of as the "blue line," is characteristic of lead poisoning. Not uncommonly a sweetish taste in the mouth is experienced. Wrist-drop as the result of affection of the musculospiral nerve is at times present in this form of intoxication.

Disturbances of the skin are impairment in sensation, usually anesthetic areas. Altered vision is occasionally met with in the form of amblyopia and amaurosis.

Examination of the blood reveals, in a large percentage of cases, basic granulation of the erythrocytes. While this condition is seen in other diseases, it is important to remember that basic granulation is rather constantly found in chronic lead poisoning. The number of erythrocytes and the amount of hemoglobin is reduced, varying with the severity of the poisoning.

TREATMENT.

Acute poisoning, unless the patient has vomited and thoroughly emptied the stomach, demands that a stomach-pump should be used. As a chemical antidote, a solution of a soluble sulphate (sodium or magnesium) should be given in full doses, so that the purgative effect may be obtained. This solution can be given directly into the stomach through the stomach-tube after the stomach has been thoroughly washed. The resulting gastro-enteritis should be treated by the application of heat to the abdomen locally; only demulcent drinks and liquids by mouth; and, if pain is intense, small doses of opium preparations.

Chronic Poisonings. Lessening the dangers in the "lead industries" will do much toward decreasing chronic plumbism. Absolute cleanliness, especially of the hands and nails, is of utmost importance. Fans, face-masks, and suitable means of ventilation should be provided wherever dust is generated. No food should be eaten in any part of the works.

Treatment should be directed toward eliminating the poisons and in relieving the immediate symptoms. Constipation should be relieved by saline cathartics, preferably Glauber or Epsom salts.

Hot packs or other means of inducing perspiration are of great aid in the elimination of lead, and at the same time relieve the kidneys of part of their work.

Potassium iodid, in doses of 5 to 15 grains (0.3 to 1 Gm.) three times a day, aids in the elimination of the lead.

Sulphur baths have been recommended as giving good results. They are prepared by mixing in a wooden tub 3 or 4 ounces (90 or 120 Gms.) of potassium sulphuret with about 20 gallons (80 l.) of water.

Colic will require hot applications to the abdomen, opiates and atropin hypodermically.

For the paralysis, strychnin, massage, and electricity are valuable means of treatment.

PHOSPHORUS POISONING.

Acute poisoning by phosphorus is not common, and usually results from the swallowing or administration of phosphorus match-heads with suicidal intent.

At the present time chronic poisoning by phosphorus is rare, and occurs in localities containing phosphorus industries.

Important changes take place in the blood and liver. The coagulability of the blood is reduced, and hemorrhages occur into the skin and mucous membranes. There is jaundice, and fatty degeneration of the viscera and muscles, especially the liver which undergoes enlargement, and changes to a bright saffron color—a type of fatty icteric liver.

The liver and blood contain intermediate products of protein metabolism such as leucin, tyrosin, and sarcolactic acid. Sugar may be found in the urine. As a result, the ammonia is greatly increased, urea decreased, and the condition of acidosis is present.

In chronic phosphorus poisoning the chief pathologic change consists of necrosis of the inferior maxilla and surrounding tissues, with suppuration and formation of sequestra. These lesions, fortunately not of common occurrence at the present day, become very extensive unless treated promptly.

Acute phosphorus poisoning bears a close resemblance to

icterus gravis and to acute yellow atrophy of the liver, from which it must be differentiated.

Soon after the poison has been ingested, vomiting and diarrhea are noted, and these symptoms tend presently to subside, only to reappear in two or three days, accompanied by jaundice, pain in the abdomen, and pains and tenderness in the muscles of the body. Fever in moderate amount is often present.

At this time blood in the vomitus and stools, petechia, and submucous hemorrhages appear. The liver enlarges in from two to four days after the onset of symptoms, and is tender. Later a decrease in size will occur if the patient recovers.

Profound asthenia, with maniacal excitement, is followed in fatal cases by stupor and coma, ending by death, which usually takes place within a week after the onset of symptoms.

If recovery takes place, the liver gradually returns to normal size, and the general condition of the subject improves, but the heart commonly shows some degree of myocardial weakness, caused by fatty degeneration.

In the chronic form of poisoning, necrosis of the jaw commonly begins about a single tooth, with inflammation and abscess formation.

Usually not only the teeth but also the surrounding tissues are affected. The pus is generally abundant and very foul. As the result of necrosis, one or several sequestra may form.

Rapid anemia and general sepsis may occur in neglected cases of either variety of poisoning.

TREATMENT.

If red phosphorus were used in the manufacture of all matches, instead of white phosphorus, poisoning by phosphorus would be practically eliminated.

The treatment of the necrosis consists of removing the subject from the danger of exposure to phosphorus, and immediate surgical and dental care.

Acute poisoning should be treated by an emetic, preferably copper sulphate, both for its emetic effect and because it prevents absorption. By means of a stomach-tube gastric lavage should be performed, using either a 2 per cent. solution of permanganate of potash or a solution of peroxid of hydrogen 1

to 3 per cent. Old spirits of turpentine has been given in doses of 0.5 mil (*7 m.*) in milk for a period of a week. Permanganate of potash, hydrogen peroxid solution, and old spirits of turpentine are used because their oxidizing properties render the phosphorus practically non-poisonous. Alkalies should be given freely, and are of value in the treatment of acidosis. The alkalies may be given by mouth, bowel or intravenous injection, if urgently needed.

COPPER, ZINC, AND TIN POISONING.

Copper. Soluble salts of copper, if taken in sufficiently large quantities, can produce acute poisoning. Chronic poisoning, by soluble salts of copper is very uncommon, as evidenced by the infrequency of such case in workers in and about this metal.

The symptoms of acute poisoning are necessarily variable, inasmuch as the soluble salts of copper in poisonous doses act as emetics. Severe gastro-enteritis, to which may be added in twenty-four hours a hemolytic jaundice, with symptoms of an acute parenchymatous nephritis, make up the picture of acute poisoning. The urine, in addition to the findings of the acute nephritis, gives a positive test for the presence of copper.

Even though the copper has been vomited, owing to its emetic action, the stomach should be thoroughly washed out with an abundance of water. Demulcent drinks, white of eggs, milk, or some albuminous substance is used to limit or prevent the corrosive action of the copper. Frequent irrigation of the bowels is of utmost importance.

Elimination should be encouraged by taking an abundance of water, and by sweating induced by hot packs or cabinet baths.

The chemical antidote is ferrocyanide of potash, which forms an insoluble salt with the copper. The irrigation of the stomach and intestines is necessary, and the administration of a chemical antidote is merely an aid in the prevention of absorption of copper.

Zinc, like copper, if given in sufficiently large doses, exerts an emetic action, and therefore its toxic effect in acute poison-

ing depends upon the size of dose and the emetic effect thereof. Recoveries from large poisonous doses may occur.

Zinc chlorid, sulphate, and sulphid are the salts commonly used in medicine and in the arts. The question of acute poisoning from the use of zinc solder used in sealing various canned goods, is so questionable that it does not warrant serious consideration. It has been found that individuals manifesting poisonous symptoms after partaking of the contents of a can in which zinc solder has been used are suffering from poisonous effects of food, rather than the effects of the zinc. The acute poisoning by zinc salts is characterized by active gastro-intestinal symptoms, and may be followed later by convulsions and epileptiform fits. Zinc chlorid locally produces a corrosive action upon the skin and mucous membranes.

Chronic poisoning by zinc is very unlikely, and the ill effects produced in the zinc industries are from the inhalation of dust of other metals, such as lead and arsenic, from a gas like carbon monoxide, or from the fumes of sulphuric and sulphurous acids.

Washing out the stomach and rest for the gastro-intestinal tract are the first indications. Thirst may be relieved by giving fluids by the rectum, or subcutaneously, if no fluid can be taken by mouth. Mucilaginous drinks should be given.

Tin. Poisoning by tin is in all probability very uncommon, inasmuch as the evidence shows that when poisoning was thought to have resulted from the container as in canned goods in tin containers, the intoxication resulted from decomposed food which had been canned.

SILVER POISONING OR ARGYRIA.

Poisoning by silver is but exceptionally always chronic in type. When taken internally, especially having been prescribed over several months or more in therapeutic doses, general argyria may result. Local argyria in those who handle silver in their occupations when seen is chiefly in the skin of the hands. The prolonged local use of silver preparations may result in local argyria. As the result of the dangers, argyria is fortunately not very common.

The pathology of argyria, briefly stated, is a deposit of silver in the skin or mucous membrane alone if in the local form. In generalized argyria, in addition to the skin and mucous membrane, scarcely a tissue of the body but that contains deposits of silver. Chronic interstitial changes, as the result of irritation produced by the deposit of silver, occur in the lungs, liver, kidneys, and spleen. The brain, its covering, and the blood-vessels are at times affected by the sclerosis produced by the silver deposits.

The principal manifestation of the disease is the pigmentation without subjective symptoms. A line is noted on the gums similar to the line in lead poisoning, but of a more decidedly violet color. The color may appear even following the discontinuance of the use of silver salts.

TREATMENT.

No known treatment will change the pigmentation for the better. Once present always present.

Silver nitrate should not be given in more than ¼-grain (0.015 Gm.) doses, and not longer than for one-month periods, with from two- to four- week intervals.

Local applications should be used, under the direction of the physician, who will see that the patient is warned against the continued use of the metal, cautioning him about the danger of chronic poisoning.

ILLUMINATING GAS POISONING.

For practical consideration, the poisonous effect produced by illuminating gas may be considered identical to that of carbon monoxid poisoning. While it has been shown, chemically and experimentally, that the effects produced by illuminating gas are not identical to those of pure carbon monoxid, nevertheless no other constituent of illuminating gas will produce so close a typical picture as that of carbon monoxid.

Gas poisoning is a method frequently used to commit suicide, probably so because of its easy accessibility in towns and cities. In addition to death produced by intentional inhalation, many deaths have resulted through improper closure

of gas jets and cocks, and through leaks in gas conduits. Gas poisoning may be acute and chronic.

Illuminating gas manufactured from coal contains 5 to 10 per cent. of carbon monoxid, while that made from wood may contain as much as 60 per cent. Water-gas, so largely made for illuminating and cooking purposes, contains approximately 30 per cent. Cases of chronic poisoning, strange as it may seem, often escape recognition because of the difference in cause and effect, as compared with acute cases. Slow leaks in gas fixtures may produce symptoms which often are not recognized as being of gas origin until it has been noticed that with the discovery and repair of gas-leaks the health of an individual or group of individuals has returned to normal. The leak need not be in the building, but may travel through the ground for some distance before entering a house. The characteristic odor of illuminating gas in such cases is usually lost.

Poisoning by combustion or incomplete combustion products such as are produced in a heating apparatus with poor draught in charcoal, iron, and other furnaces, and in the coal-tar industries, is due chiefly to the effects of carbon monoxid. The poisonous effects produced by the use of the gas- and oil- stoves in small and poorly ventilated rooms are striking, and should be guarded against.

In acute cases of poisoning, the striking changes produced are widespread in the body. The deadly effects of carbon monoxid are due to the readiness with which the gas combines with the hemoglobin, and to the stability of the compound thus formed. The skin is bluish or bluish-red as the result of extravasations beneath the skin. The body presents a mottled appearance. The muscles show degenerative changes, and scarcely an organ or tissue escapes the hyperemia and scattered small hemorrhages. The respiratory tract may present a bronchitis, or bronchopneumonia; rarely a lobar pneumonia develops. Quite commonly the kidneys show acute parenchymatous changes. Cerebral symptoms may be present, owing to the multiple scattered hemorrhages throughout the brain tissue.

In chronic poisoning the fatty changes in the cardiovascular system are noted. Marked anemia is due, in part, to the destruc-

tion of the erythrocytes, and in part to the failure of kidney action.

The combination formed by the gas and hemoglobin is a staple one; that is, the hemoglobin no longer carries oxygen, and as a result the tissues suffer asphyxiation. With proper treatment individuals severely poisoned, and apparently fatally ill, may recover.

The symptoms of acute gas poisoning have been variously described by those who have recovered. The clinical picture ordinarily is that of severe headache with early a throbbing of the blood-vessels, accompanied by a burning sensation in the face, nausea and vomiting, and attacks of vertigo, with rapid oncoming muscular weakness. The subject becomes drowsy, and slowly passes into an unconscious state. Muscular twitchings are often alone present or associated with convulsions.

It must not be forgotten that frequently a poison is taken by mouth with suicidal intent, and, in order to accomplish the individual's object, he further inhales gas, so that the clinical picture that is presented to the physician is a double one. This fact must be borne in mind in the treatment of what appears to be a gas poisoning case with unusual symptoms.

Drawn blood presents the cherry-red color, and gives the chemical and spectroscopic tests for carbon monoxid. Convalescence may be very slow, and a confused mental state may at times entirely escape recognition.

The sequelæ of the acute poisoning deserve special mention. The respiratory tract may be affected to the extent of pneumonia. Nephritis may be acute, only to end in a chronic form.

The heart and blood-vessels seldom escape without more or less permanent sequelæ. Irregular heart or palpitation often occurs for an indefinite period afterward.

Probably the nervous system is implicated more markedly than any other system. The various paralyses, choreiform movements, anesthesias, neuralgias, tremors, alteration of speech, have been observed. Changes in the special senses, such as deafness of different grades, associated with roaring noises in the ears, occur; various ocular disorders are not very common.

The mental changes vary in type, and include simple confusion, delirium, mild or noisy, and hallucinations; simple dementia may persist.

Chronic poisoning may be difficult to distinguish, or it may at times entirely escape recognition. A history of gas leaks, or workers about the gas industries and associated therewith, with headache, nausea, vomiting, poor health, mental disturbances, lack of concentration, sluggish intellectual action, make up a picture of chronic gas poisoning.

TREATMENT.

The treatment of acute poisoning consists of prompt removal of the subject from the gas-laden atmosphere. Oxygen should be freely supplied. To what extent oxygen in excess of the amount present in the fresh air is absorbed is a much disputed question.

Artificial respiration and various mechanical devices, such as pulmotor, lungmotor, are used in resuscitation.

Venesection, promptly followed by saline infusion, or direct transfusion of blood, is a life-saving measure.

The circulation may require stimulation by digitalis, strychnin, caffein or atropin in dosage necessary to produce prompt results. External heat is indicated if the temperature has dropped below normal.

The patient should be carefully watched, since he may at any time (if the gas has been taken with suicidal intent) further seek means to accomplish his purpose. The fact also must not be lost sight of that the patient may be suffering from the effect of another poison in addition to gas, *e.g.*, bichlorid of mercury or acute alcoholism. Tonics are a necessary detail in the after-treatment of this sort of cases.

COCAINISM.

While therapists are divided with regard to the desirability of withdrawing morphin gradually or suddenly, they are in more general accord in advising the immediate withdrawal of cocain in cases of chronic poisoning by this drug. The symptoms of immediate withdrawal, such as insomnia, palpitation, dyspnea and collapse, are often quite as distressing as in morphinism, but are more readily controlled. The success of treatment in any given case is dependent upon the presence or absence of a previous neuropathy, the circum-

stances under which the habit was contracted, the length of time it has existed, and the association with other drug addictions, such as morphin and alcohol. In some instances it is the indirect outcome of nasal surgery (Dercum). Many nasal applications contain cocain, and their use may cause the cocain habit, but in most cases it is acquired by morphin habitues who go to cocain in the expectation of finding help in their struggle against the tyranny of the former drug. In this hope, however, they are always disappointed when the drugs are in their own hands. The victim soon finds that one of these agents antagonizes the other to a great extent, while, at the same time, it sets up peculiar troubles of its own; and that there is a constant need of more morphin to counteract the cocain symptoms, and of more cocain to antagonize the symptoms due to the increased amount of morphin. The result is that one who is using only a moderate daily amount of morphin, if cocain be added, will soon be taking a great amount of morphin, as well as of cocain, and "the last state of that man is worse than the first." Drug fiends who use both drugs frequently state that cocain is not a habit in the sense applied to morphin. The latter when once started requires daily use; cocain, however, is used more for periodic debauches, known by the Tenderloin dweller as "coke sprees" and "coke parties," in the intervals of which there may be little or no craving for the drug. Cocain therefore can be removed promptly by treatment, and with less suffering than in the case of morphin.

TREATMENT.

In treating cocainism we must realize a total irresponsibility on the part of the patient, and that no effort, short of placing him in an institution, isolated from everyone except his physician and two trusted attendants who can carry out every detail of treatment, will avail. The most effective treatment is known as the *Towns-Lambert treatment,* and is the same as that applied for alcoholism, except that no cocain is given at any time, and that strychnin or some such stimulant must be given from the beginning. (See p. 439.) The mixture of belladonna—xanthoxylum and hyoscyamus (tincture of belladonna [15 per cent.], 2 parts; fluidextracts of xan-

thoxylum and hyoscyamus, each 1 part)—with 5 grains (0.33 Gm.) of blue mass and 5 compound cathartic pills, are given simultaneously at the first dose. The belladonna mixture is continued every hour of the day and every hour of the night, the same as with the morphin patients, and twelve hours before the initial dose patients are again given from 3 to 5 compound cathartic pills, and at the twenty-fourth hour after the initial dose they are again given the cathartics, followed by salines if necessary, and again at the thirty-sixth hour. After these last cathartics, the bilious stools will appear, and by the forty-fourth or forty-fifth hour the castor oil is given. Sometimes it is necessary to carry on the treatment over another period, and the compound cathartic pills and blue mass are again given at the forty-eighth hour. It may even be necessary to carry on the treatment one or two periods longer. If these patients are excessively nervous it is necessary also to see that they sleep, and the mixture of chloral hydrate, 20 grains (1.3 Gm.); morphin, ⅛ grain (0.0082 Gm.); tincture of hyoscyamus, ½ dram (7.5 mils); tincture of ginger, 10 minims (0.67 mil); tincture of capsicum, 5 minims (0.33 mil); and water, ½ ounce (15 mils), is the best hypnotic for them. These patients should also have cardiac stimulants such as strychnin and digitalis after the first twenty-four hours, or sooner if they are weak.

The result of this treatment is a complete obliteration of the terrible craving that these patients suffer, and it is infinitely superior to gradual withdrawal of the drug. As a rule the active treatment so instituted does not cover a period of more than two or three weeks at the most. Adjuvant treatment resolves itself into improving the patient's mental attitude and general physical condition. Hydrotherapy, massage, gentle exercise, nutritious food, an abundance of rest, and all that makes for a healthful and invigorating environment should be placed at the disposal of the patient. For sleeplessness the bromids are very efficacious, and in some instances moderate doses of trional or sulphonal.

The difficulty in all these cases lies in the fact that, although they have been freed from the desire, the continuance of the cure is largely in their own hands, and a return to the former environment and habits of life leads to relapses.

The problem then becomes one of mental regeneration, which is, perhaps, the most difficult phase of the treatment.

When, as a result of profound intoxication, the patient has passed beyond the legal boundary of sanity he can be committed and restrained in an institution; but, as Dercum states, it is unfortunate, to say the least, that we must frequently wait until gross insanity supervenes before effective treatment can be instituted.

OPIUM POISONING.

The recognition of *acute opium poisoning* is based partly upon the history and partly upon symptoms, such as slow, stertorous breathing, livid cyanosis, contracted pupils, warm and dry skin, cold sweats, slow, full pulse, and drowsiness, if not deep sleep.

TREATMENT.

Gastric lavage should be promptly resorted to, unless emesis has been very thorough. This procedure is indicated regardless of the time that has elapsed since the opium or any of its preparations have been introduced into the body. It is necessary that gastric lavage be practised early, because it has been shown experimentally on animals that morphin given subcutaneously can be recovered from the stomach; if allowed to remain in the stomach it may be reabsorbed by the gastric mucosa. A 1:500 solution of permanganate of potash has proved most efficacious for use in gastric lavage. Several ounces should be permitted to remain in the stomach after the lavage has been completed, in order thus to neutralize the opium eliminated into the stomach.

The patient should receive at intervals 4 to 6 ounces (120 to 180 mils) of warm coffee by the rectum. Artificial respiration should be kept up if the skin indicates too great depression of the respiratory center.

Strychnin hypodermically, $\frac{1}{30}$ to $\frac{1}{10}$ grain (0.002 to 0.006 Gm.) is most valuable in combating respiratory failure, and it exerts a stimulating effect upon the nervous system. Atropin sulphate in doses of $\frac{1}{100}$ to $\frac{1}{50}$ grain (0.0006 to 0.0013 Gm.) is used for a similar purpose, but not as successfully.

If the patient can swallow, the administration of stimulants by the mouth, such as aromatic spirits of ammonia or whisky, may be resorted to advantageously.

Since every effort should be directed to keep up the activity of a paralyzed respiratory center, patients are frequently lashed with wet sheets or towels, or walked about, even if in an exhausted condition. This procedure should be used only as a last resort, and in the absence of a better method, which consists in the application of the full force current of an ordinary medical battery. The two small poles wet with salt solution, or, better still, a wire electric brush, should be swept over the body, while the negative pole is held in the hand of the patient or pressed against the skin (Hare).

In the advanced stage external heat is necessary to maintain body temperature.

Treatment of Morphinism or the Morphin Habit. The opium habit consists of the introduction into the body of various alkaloids or preparations of opium, or containing opium. Morphin seems to be the most frequently used alkaloid of opium; codein, heroin, somewhat less so. Regardless of the form in which the patient takes the drug, the treatment is practically the same.

Many of the reputed and extensively advertised remedies for the cure of the opium habit have been shown to consist largely, if not entirely, of some form of opium. As a result of an active campaign by the Federal agents and the enactment of the Federal Anti-Narcotic Law, this practice has been almost annihilated.

The addicts of this habit long to free themselves from the slavery of the habit, but it requires great will-power to give the co-operation which is so essential to successful treatment.

Special institutions and wards are best adapted for carrying out successfully the treatment for morphinism. The principles governing the method of treatment are: rapid elimination of the drug from the body, and the control of the symptoms resulting from withholding the drug, with a drug of the belladonna group, and lessening the amount of opium taken as rapidly as conditions will permit.

Numerous treatments, differing however in details, have been successfully used.

It is imperative to isolate the patient from his friends, and to be sure that the attendants are trustworthy, thereby being assured that the patient will receive no drug except upon the physician's orders.

The writer has known a patient, using considerable quantities of morphin, who put himself under treatment for morphinism, and in spite of rapid withdrawal of the drug no symptoms were complained of as the result of withholding the morphin. An investigation was instituted to ascertain the source of the supply of the drug which the attending physician felt confident the patient was receiving. Finally it developed that, at the request of the patient, who was permitted to receive eggs from a certain shop in which a friend of his was employed, this friend injected morphin by means of a hypodermic syringe into each egg, thereby keeping the patient very comfortable on his morphin supply. When eggs were no longer obtained from this source an immediate change was noted.

Belladonna and hyoscyamus are used to the point of dry throat and the stage of dilated pupils with maintained consciousness and freedom of action. The prescription used is composed as follows:

Tincture of belladonna 15%	2 parts.
Fluidextract hyoscyamus	1 part.
Fluidextract xanthoxylum	1 part.

This mixture must be used until the full effect of the belladonna is obtained, and of course the maximum amount must vary with the patient.

Towns' treatment is well known as a successful method of curing chronic opium poisoning.

The details of treatment as stated by Lambert are as follows: The patient is given 5 compound cathartic pills U. S. P. and 5 grains (0.32 Gm.) of blue mass. If at the end of six hours no action of the bowels has resulted, a saline is administered. After thorough action of the bowels has been obtained, in three divided doses, at one-half hour intervals, two-thirds or three-fourths of the total daily dose to which the patient has been accustomed is given.

The larger amount of the drug prevents the disturbance to the patient that would otherwise be experienced if the dose

were very much lessened. After the second dose of morphin has been given, observe the effect upon the patient, since this quantity should be sufficient to keep the patient comfortable; in fact some patients cannot take with ease the third dose of morphin. The belladonna mixture in 6-drop doses (0.4 mil) is given every hour for six doses. At the end of six hours the dose is increased to 8 drops (0.5 mil), and dosage is increased every six hours until 16 drops (1.0 mil) are taken, when it is continued, as heretofore, hourly as the fixed dosage.

If the patient shows excessive belladonna symptoms, it is diminished or discontinued. Widely dilated pupils, excessively dry throat, erythema of the skin, or a peculiar incisive and insistent voice, and an insistence on one or two ideas, indicate that the belladonna medication must be reduced or discontinued. After the symptoms have subsided, the belladonna mixture is begun at a reduced dosage, and gradually increased to the point of tolerance.

Ten hours after the initial dose of morphin is given, 3 to 5 compound cathartic pills and 5 grains (0.32 Gm.) of blue mass are repeated, followed by a saline in six hours, unless the bowels have been thoroughly evacuated. After the bowels have acted satisfactorily the second dose of morphin is given, which is usually about the eighteenth hour. This should be one-half the original dose, *i.e.*, one-third or three-eighths of the original twenty-four-hour daily dose. The belladonna mixture is still continued, and ten hours after the second dose of morphin, *i.e.*, about the twenty-eighth hour, 3 to 5 compound cathartic pills are given again, and 5 grains (0.32 Gm.) of blue mass; if necessary, six or eight hours later a saline is given.

When a thorough action of the bowels has again been obtained, at about the thirty-sixth hour, a third dose of morphin is given, which is one-sixth or three-sixteenths of the original dose. This is usually the last dose of morphin that is necessary.

Again, ten hours after this third dose of morphin, *i.e.*, the forty-sixth hour, 3 to 5 cathartic pills and 5 grains (0.32 Gm.) of blue mass are again repeated, followed in seven or eight hours by a saline, and by this time the stools should appear green.

After the stools have become green, and the bowels thoroughly evacuated, about eighteen hours after the third dose of morphin, 2 ounces (60 mils) of castor oil should be given. It may be necessary to continue the belladonna mixture for one or two further cathartic periods before the green stools appear. During this last period, when the bowels are moving, and before the castor oil is given, the patient suffers the greatest discomfort. The nervousness and discomfort can be controlled by codein in 3- (0.195 Gm.) to 5- grain (0.32 Gm.) doses hypodermically.

About the thirtieth hour these patients should be given strychnin or digitalis, or both. Later, tonics, such as iron, arsenic, or phosphorus, are indicated. During this treatment light nourishing food is given. After the system is rid of the morphin there is danger of the patient over-eating, thus bringing back symptoms similar to those of the withdrawal period, and due to indigestion. The patient naturally assigns the cause of these symptoms to the discontinuance of the drug, rather than to the true cause, namely, that of over-eating.

If, as occurs at times, about the thirty-sixth hour, the stools become clay-colored, some form of ox-gall in small doses, repeated every two or three hours, is effective in bringing about the free flow of bile. At times it may be necessary to continue the morphin through the fourth period.

Codein and dionin are preferable to heroin to act as carrying-over drugs. If the patient shows an idiosyncrasy for one form of opium, another salt should be tried.

The after-treatment is very important. This interval should be fully occupied, preferably in the open air, with plenty of exercise, if not with a course of physical training. Unless this is done the patient may become a neurasthenic or drift back into the old habits.

The mental state of the patient will subsequently have to be improved, otherwise he will be readily discouraged. Exercise and encouragement will soon change the depressed and irritable patient, under treatment for the opium habit, to one with good spirits and normal health.

ALCOHOLISM.

The treatment of alcoholic subjects must be considered under the three separate headings of Acute Alcoholism, Chronic Alcoholism, and Alcoholism with Symptoms of Delirium Tremens.

When alcohol in some form has been consumed for the first time by those unaccustomed to its use it not infrequently produces alarming symptoms. Severe nausea, retching and vomiting, diarrhea, and finally collapse or disordered mental conditions are prominent symptoms, directly due to the effects of alcohol. During the subsidence of the attack, gastritis or nephritis may complicate the condition. Alcoholic beverages have been taken accidentally by children, who as a result have suffered severe attacks of acute alcoholism.

TREATMENT OF ACUTE ALCOHOLISM.

All efforts are directed toward the elimination of the alcohol. An emetic by mouth, or apomorphin hypodermically, is indicated, especially if the patient is too ill to have gastric lavage. The bowels should be thoroughly evacuated, either by calomel in divided doses or by an enema. The skin should be rendered active by bathing, sweating, and the ingestion of large quantities of water.

Sleep and quiet should be obtained for the patient, using chloral and the bromids, veronal or trional in full doses. It may be necessary to resort to the use of morphin hypodermically, or to an opium suppository.

The diet should be light and nourishing, preferably milk and broth, until the acute inflammatory condition of the gastro-intestinal tract has subsided. Alcoholic beverages are contraindicated. If depression is marked, hypodermic injections of strychnin sulphate, to be repeated as conditions warrant, are useful.

TREATMENT OF CHRONIC ALCOHOLISM.

This very familiar condition in some form is a problem constantly confronting physicians, police departments, and civil courts. Slowly public opinion is awakening to the fact that drunkenness is often but an incident in a life of misfor-

tune, and that jail and punishment are not remedies that will cure this type of slow poisoning by alcohol.

The treatment should be directed toward the elimination of the poison, and discovering and solving the underlying social problem that presents itself.

Lambert has found the following treatment very satisfactory:—

℞ Tincturæ belladonnæ (15%) .... f℥ij (7.50 mils).
Fluidextracti hyoscyami,
Fluidextracti xanthoxyli .....āā f℥j (3.75 mils).

This mixture is given every hour of the day and night beginning with 6-drop doses (0.4 mil) and increasing 2 drops (0.13 mil) at the end of every six hours until the patient is taking 16 drops (1.0 mil), or until dryness of the throat, dilation of the pupils, or a belladonna rash shows that the patient has reached the limits of tolerance.

Some patients can take but little of this mixture, 1 or 2 drops (0.07 to 0.13 mil) every hour, but as long as they obtain the full physiologic effects, as judged by symptoms of belladonna intolerance, the desired result is achieved.

With the first 6 drops (0.4 mil) of this mixture from 2 to 5 compound cathartic pills and 5 grains (0.32 Gm.) of blue mass are also given. Five to six hours later a saline is given. At about the eighteenth to the twentieth dose of the belladonna mixture there are also given 3 to 5 compound cathartic pills, with 5 grains (0.32 Gm.) of blue mass, followed by a saline five hours later; and again at about the thirtieth dose and the forty-second dose the pills and the blue mass are again repeated.

Often, when these last cathartics act, green bilious stools will appear, and 1 or 2 ounces (30 to 60 mils) of castor oil should be given, and the treatment stopped. Sometimes it is necessary to push the belladonna higher than 16 drops to get the symptoms of full tolerance, and to obtain the biliary reaction.

At times it is necessary to carry this belladonna treatment over the sixtieth or even into the seventieth hour, with extra dosage of cathartics every twelve hours as above.

In young and vigorous patients, the alcohol can be immediately withdrawn. As a general rule, to the older and

nervous patients, who have been on a prolonged debauch, it is necessary to give 2 ounces (60 mils) of whisky four or five times during the first twenty-four hours. Strychnin and digitalis are indicated in this class of cases. Chloral hydrate and morphin are useful in producing sleep.

If the patient has alcoholic gastritis, is nauseated and cannot retain his medicine, 10 to 20 grains (0.66 to 1.3 Gm.) of sodium bicarbonate or sodium citrate every hour for five or six doses has been very useful. In severer cases, the addition of 5 grains (0.32 Gm.) of Tully's powder (pulvis morphinæ compositus) at four-hour intervals, for two or three doses, is beneficial. Often the sodium citrate can be given with 10 to 20 grains (0.66 to 1.3 Gm.) of cerium oxalate some hours after the Tully's powder has been discontinued.

If delirium tremens develops during or following the treatment, it may be necessary to keep the patient asleep with hypnotics, giving cathartics once in twenty-four hours, and abundant nourishing food, as milk and eggs.

If the milk does not agree with patients who are taking the belladonna mixture, a light diet of eggs, broths, bread and butter, or a small amount of meat and vegetables can be substituted.

Chronic alcoholism is being treated in various private institutions throughout the country more or less upon the principles governing the foregoing treatment. Unfortunately, the poorer classes, who suffer from the continued effects of alcoholic poisoning, are in no position to receive this thorough treatment. It is incumbent upon the public to establish, in connection with municipal hospitals, provision for the treatment of chronic alcoholics, as those suffering from disease and not as criminals.

TREATMENT OF DELIRIUM TREMENS.

Delirium tremens is a condition brought about as the result of chronic alcoholism. It is not dependent upon the amount of alcohol taken, as many men who have never been intoxicated, but who have been continuous users of alcohol for years, when confined to bed on account of severe illness, or accident, have suddenly developed mania à potu.

The treatment is largely symptomatic. The prognosis is dependent upon the condition of the various organs of the body, and upon the severity of the associated condition, such as an acute infection or fracture. A subject of chronic alcoholism usually shows disease of the cardiovascular and the renal system, and treatment is aimed at rendering as efficient as possible these vital organs.

Authorities differ as to the withdrawal of alcohol in victims of delirium tremens, but as a general rule it is advisable absolutely to forbid alcohol in the young and healthy patient, and in the weak and elderly to reduce slowly the amount taken.

The supportive measures are of first importance. The degenerated heart-muscle should be stimulated with hypodermics of strychnin, caffein, or digitalis.

Elimination by means of vigorous purgatives must be obtained by the administration of compound cathartic pills, blue mass, calomel, or a pill containing calomel powder, squills, and powdered digitalis, of each 1 grain (0.065 Gm.).

The skin should be rendered active by placing the patient in a warm bath; if delirious, this form of hydrotherapy continued for several hours under constant supervision, affords a great deal of relief.

An abundance of water should be given by mouth, by bowel, or by hypodermoclysis in the form of salt solution, for by this procedure the toxemia is decreased, and the skin and kidneys are stimulated to greater activity.

Emesis is contraindicated in the feeble and elderly, and in the state of delirium tremens it is of questionable value.

Often in the severest cases of young vigorous individuals apomorphin hypodermically $\frac{1}{10}$ grain (0.0065 Gm.) produces vomiting early, followed by a period of quiet, if not sleep.

Rest for the patient is extremely difficult to obtain. Paraldehyd, in doses of from 1 to 2 drams, (4 to 8 mils), to be repeated in an hour, is valuable in producing a sleep, from which the patient may awaken either refreshed and clear-minded or with the delirium much lessened.

Chloral trional, veronal, sulphonal, medinal, and other hypnotics are useful in mild cases. Hyoscin $\frac{1}{100}$ grain (0.00065 Gm.) and morphin have to be resorted to, to secure quiet in those with motor symptoms.

Chloral and morphin combined act very efficaciously. A combination of chloral and the bromids are useful in mild cases.

Hypnotics do not necessarily cut short the delirium, but they act by securing rest for the patient.

Lambert has used with success ergot hypodermically in Livingston's solution, which is as follows: One dram (3.9 Gms.) of solid extract of ergot is dissolved in an ounce (30 mils) of sterile water, and 3 drops (0.2 mil) of chloroform and 3 grains (0.195 Gm.) of chloretone are added, and the solution filtered; this is sterile, and can be given directly into the muscles in gluteal region or into the deltoid.

It should not be given subcutaneously, as it produces great pain. The administration every two to four hours reduces the dilated blood-vessels, lessens the various congestions, and brings about a better equilibrium of the circulation. After its administration, there is a tendency to a quieter delirium, and less need of restraint.

As soon as possible nourishing food should be given in the form of milk, eggs and milk, or broths.

The patient should be confined to bed while in a delirious state. A sheet folded and extending across the chest, with the wrist and ankle shackles, is a better means of restraint than a straight, rigid jacket extending the greater length of the body, and thereby preventing the radiation of heat.

During convalescence, bitter tonics are indicated in the form of capsicum, gentian, and nux vomica.

Exercise in moderation, fresh air, and the proper amount of sleep are essential in the restoration to health, which cannot be complete without total abstinence from alcohol.

Methyl Alcohol. Wood alcohol, Columbian, colonial, union or eagle spirits.

The ingestion of 2 drams (7.5 mils) has been followed by blindness; in other individuals 1 ounce (30 mils) produced only intoxication. Another curious condition is that symptoms may be delayed twenty-four hours.

The general treatment consists of prompt gastric lavage, vigorous stimulation by strychnin, camphor, and digitalis hypodermically, saline irrigation by the bowel, free sweating and the use of emetics.

The optic nerve atrophy, which is a common result in those convalescent from the effects of methyl alcohol poisoning, is treated by pilocarpin, sweat-baths, and the use of iodids, although recovery from the grave accident cannot be counted upon in many instances. Strychnin in large doses later seems to limit the extension of secondary optic nerve atrophy.

FOOD POISONING.

The success of the treatment in food poisoning must necessarily depend here, as in all conditions, upon the recognition of the cause.

Certain general principles govern, and are applicable to, all forms of food poisoning. It is very difficult at times to determine the cause of an illness, as to whether the origin lies in poisoned food, or is due to bacterial infections and intoxications.

The treatment consists in the use of prophylactic, symptomatic and eliminative measures.

Prophylactic measures, such as the inspection of cattle for diseased conditions prior to killing, examination of the meat after it is dressed, inquiry into the sanitary conditions of the slaughter-house and its employees, and the subsequent storing and preparation for eating, are the best safeguards against meat poisoning.

Bacteria may be destroyed in the process of cooking, but this, of course, cannot be relied upon to destroy products of chemical changes.

Abdominal pain is usually a prominent symptom of ptomain poisoning. Locally, heat or cold, preferably the former, and counter-irritation by means of a turpentine stupe or a mustard plaster are useful. After the bowels have been thoroughly emptied by castor oil, or a mercurial followed by a saline, if the abdominal pain persists, small doses of an opiate may be required.

High colonic irrigation with warm normal salt solution, allowing 6 to 8 ounces (180 to 240 mils) to remain in the colon, should be given in every case with more than moderate severe symptoms.

If considerable retching and vomiting occur, washing out the stomach tends to relieve this symptom.

Food should be restricted for at least twenty-four or forty-eight hours following the onset of symptoms, and with the beginning of improvement of the patient's condition liquid diet in small quantities, at short intervals, can be given. Usually the patient does not complain of the lack of food. The most suitable foods are milk and albumin water, increasing to semi-solid food as the gastro-intestinal symptoms subside.

If the patient is very much prostrated, or if collapse occurs during the acute symptoms, the use of stimulants, such as atropin, strychnin, and aromatic spirits of ammonia is indicated.

The serums prepared as antidotes for bacterial infection of food, particularly for meats, have been used, but not with a great degree of success.

SUNSTROKE.

(Thermic Fever, Insolation, Heat Exhaustion.)

The treatment of conditions arising from the exposure of the human body to too great a degree of heat, regardless of the source, is entirely preventible when rigid prophylactic measures are practised. Humidity is an important factor in intensifying the effects of heat.

Prophylaxis. In the larger cities the housing conditions and sanitation have helped in a measure to reduce the mortality from heatstroke, especially among the very young and the aged. A further degree of success has been attained by moving the city children to the country and seashore, so as to enjoy the better air of such environments. The value of public parks and city squares is being more appreciated for the benefit of children during the hot weather.

In countries with a more or less uniform hot climate the number of fatalities from the heat is not so great as in the United States, where temperature variations are extreme. People have learned the value of prophylaxis. Public demonstrations and gatherings are avoided during the warm hours of the day. Marches and parades are dispensed with, and cool spots are sought. All living and working rooms should be well ventilated, if necessary artificially changing the air,

which can also be cooled by such a means. Every effort should be directed to lessen the individual's exposure, and to increase his resistance to the heat.

Proper clothing is essential, if exposure to the heat is necessary. Suitably fitting straw hats, well ventilated, and constructed so that moist sponges or cloths may be carried therein, should be worn. Paradoxical as it may seem, flannel, light in weight, is the best material for summer wear. This material protects from extreme heat, and at the same time increases the radiation of body heat. Clothing should be loose fitting. Bathing should be frequent, for the sake of cleanliness and for stimulatory effect upon the body, which counteracts the depression resulting from excessive degrees of heat.

Moderation in all things is well applied to the mode of living in hot weather. Alcohol is to be avoided, except for those to whom because of long usage it is a necessity.

Physical or mental work should be limited. As radiation progresses much fluid is lost by perspiration, and it is essential that large quantities of fluid be taken to replace this loss. Ice-cooled water is better than iced water. Fruit juices may be added to carbonated water, and served as a good substitute for plain water. Iced tea and coffee are recommended for those taking these beverages daily.

The diet should be moderate in amount, with small amounts of carbohydrate and fat. Fruit and vegetables in abundance are most useful.

Constipation must be guarded against by taking plenty of water, fruits, vegetables and laxatives if necessary. Diarrhea calls for physical rest, and the limitation of or abstinence from food for a short period.

TREATMENT.

The symptoms divide the patients into two classes: first, those with *thermic fever,* usually hyperpyrexia; and second, *heat exhaustion* (subnormal temperature), and presenting symptoms referable to the nervous system.

In the condition of hyperpyrexia the indication is to reduce without delay the temperature, which in itself is fatal if not controlled.

For this purpose, ice water enemata, ice rubs, ice water tub baths, and ice packs are used. The method most readily available should be used. Drugs that increase cerebral congestion and those that stimulate the functions of the body are contraindicated. If hyperpyrexia does not exist, the milder hydrotherapeutic measures may be applied. In all forms of treatment directed toward lowering the temperature, it is important to take the temperature by rectum during the treatment. This temperature must not be allowed to become lower than 100° F. (37.8° C.), and treatment must be discontinued when this point is approached.

The cases of heat exhaustion referred to as being characterized by nervous symptoms are treated by stimulants. Strychnin, caffein, and atropin are to be given hypodermically for their effects on the vital centers, vasomotor, cardiac and respiratory. Rapidly acting diffusible stimulants by mouth, such as alcohol, aromatic spirits of ammonia, spirits of camphor, and Hoffman's anodyne have a rapid beneficial effect. Oxygen inhalation and artificial respiration are indicated if symptoms of respiratory failure are manifest.

If dilatation of right heart is present, venesection is demanded to relieve the venous congestion consequent to this emergency. Light massage of the extremities aids in equalizing the distribution of the blood. If the temperature continues subnormal, external heat is indicated.

Although a patient may survive a severe attack of sunstroke, in a large percentage of cases the subject is never restored to his original health.

Intolerance of heat, even a mild degree thereof, is a common after-complaint, and removal of the patient to a cooler climate is indicated. Frequently various cerebral conditions are complained of, and physical disturbances, such as loss of memory, irritability, insomnia, mental hebetude, and dementia are not uncommon.

Convalescents should be closely guarded in order to prevent suicidal attacks. The after-care is difficult, and presents conditions not unlike those noted in illuminating-gas poisoning (*q.v.*).

Diseases of Metabolism and Nutrition

BY

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Diseases of Metabolism and Nutrition.

FOREWORD.

In the section on Diseases of Metabolism an endeavor has been made to embody the fundamental facts and theories in a preliminary chapter to which have been appended useful dietetic tables. In the articles on the individual diseases the etiology, physiologic chemistry and symptoms have been considered with sufficient fullness to serve as a guide to treatment. The medicinal treatment of these affections offers little that is novel, so that the effort has been to make a rational selection of well-known remedies. The prophylactic treatment and general management have been more fully considered, and no pains have been spared to make the dietetic treatment as explicit as possible. In Obesity caloric diets have been worked out, while in Diabetes the useful tables of Janeway and Joslin (Allen treatment) have been introduced. In the discussion of Scurvy the importance of vitamins has been emphasized; while under the caption of Chronic Arthritis, the newer dietetic methods have been explained.

GENERAL CONSIDERATIONS.

The intelligent treatment of the constitutional affections requires not only a knowledge of the pathology and symptomatology of the various diseases and syndromes, but also a clear conception of the underlying principles of metabolism.[1] It will be convenient to discuss the latter division of our subject in a preliminary chapter. This will allow of a briefer discussion of chemical pathology in the chapters on the individual diseases. It will also permit us to introduce a few observations on general dietetics.

Definition. Metabolism is the name applied to those physical and chemical processes occurring within the living body, by means of which heat and energy are liberated, and nutriment is assimilated and built up into living structures; or, on the other hand, effete tissues and waste products are broken down and excreted from the body. The term is not applicable to changes in the food, which occur in the stomach and intestines before absorption, or to alterations in the secretions and excretions after

they have escaped from the glands of the skin, kidney and gastrointestinal tract, or from the alveolar epithelium of the lung.*[2]

Proteins. The principal substances which furnish heat and energy are the proteins, hydrocarbons and carbohydrates. Proteins also serve to renew the body cells, while fat and glycogen are stored as an energy reserve. In addition to these principal foodstuffs there are important mineral salts and peculiar substances known as vitamins. Water and oxygen, although not foodstuffs, are of equal importance in the chemical processes, and are utilized, the latter particularly, in definite or even quantitative amounts. The proteins differ from the other important foodstuffs, in that they invariably contain nitrogen in addition to carbon, hydrogen and oxygen. The various proteins also contain other important elements, such as iron, sulphur, phosphorus and iodin. Chemically they are very complex substances, frequently containing a large number of atoms in the molecule. As these molecules are capable of an almost infinite variation in their internal arrangement, the possible combinations are enormous. According to the modern conception, each protein molecule is made up of a comparatively large number of complicated substances, known as amino-acids, loosely bound together. During digestion and assimilation, proteins are broken down into their constituent amino-acids, and are afterward built up into the particular forms required by the individual. It therefore makes little difference in what form protein is ingested, provided a sufficient number of amino-acids are furnished. Gelatin, which closely resembles protein, is inefficient as a foodstuff, because it lacks certain essential amino-acids. The proteins of meat, fish, eggs and milk are similarly superior to those of wheat, beans and Indian corn. A wide selection of vegetable proteins will, however, furnish all the necessary elements of the diet. The amino-acids are often spoken of as "building stones," probably in allusion to the toy building stones with which such a multiplicity of structures can be erected. The principal "building stones" are glycocoll, alanin, valin, leucin, prolin, phenylalanin, aspartic acid, glutamic acid, serin, cystin, tyrosin, lysin, histidin, arginin and tryptophan. The daily requirement of protein is approximately 1 gram (15 grs.) for each kilogram (2.2 lbs.) of body weight.† This, according to Tay-

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* Slightly modified from Farr: Medicine for Nurses, Philadelphia, 1915.

† It is convenient to use the metric system in estimating calories even if we afterwards convert our results into ordinary measures.

lor,[3] gives 100 per cent. margin, although at least 50 per cent. less than the old empiric standards, which were based on a study of the diets actually employed by Europeans. A still further reduction is admissible, if necessitated by impairment of the excretory functions.

Only a small amount of the protein is actually used for growth and reconstruction; the remainder is split up into sugar and a nitrogenous fraction, and thus becomes available for the production of energy. Most of the nitrogen is eliminated as urea, which still has a certain caloric value, so that the heat value of protein, which is theoretically 5.7, is actually only 4. The utilization of protein seems, in itself, to require a large amount of energy, so that at least 25 per cent. of the remaining calories are lost, *i.e.*, they are given off as external heat (specific dynamic factor of protein). In contrast to protein, carbohydrates and hydrocarbons are completely oxidized to CO_2 and H_2O, and eliminated through the lungs. The specific dynamic factor for carbohydrates is stated as 6 per cent., and for hydrocarbons as 12 per cent. (Taylor). It is readily seen, therefore, that protein is not an economic food for the production of heat and energy.

The major portion (approximately 80 per cent.) of the nitrogen elimination in the urine is in the form of urea, the remainder appearing as creatinin, uric acid and purins. The creatinin excretion varies directly in proportion to the catabolism of muscular tissue, and is practically a fixed amount for each individual. The uric acid and purins are also dependent on a special form of catabolism, that of nucleic acid. They are derived from the breaking down of the nuclei of the fixed and wandering cells (leucocytes) of the body (endogenous fraction), or from the catabolism of similar cells introduced with the food (exogenous fraction); the latter fraction can be eliminated by a purin-free diet, the former cannot.

These nitrogenous substances, including urea and the other non-protein bodies, are carried to the kidneys, and there largely eliminated. The ingestion of nitrogen in the food is almost balanced by its excretion in the urine. The difference, which amounts to 1 or 2 grams (15 or 30 grs.), is eliminated to a slight degree through the skin, but more largely through the intestines. The blood ordinarily contains 25 to 30 milligrams of total non-protein nitrogen per 100 mils.* [4] Approximately one-half of this

* In nephritis, with or without uremia, the total non-protein nitrogen may be enormously increased (70, 150, 180 milligrams or more per 100 mils).

amount consists of urea. The balance comprises, in addition to excretory products, newly absorbed amino-acids on their way to utilization. Normal blood (purin-free diet) contains from 0.5 to 2.9 milligrams of uric acid per 100 grams, or an average of 1.4 milligrams.* In the early part of an attack of gout this amount may be considerably increased, while subsequent to the attack it may reach subnormal value.

Hydrocarbons. The hydrocarbons, which include the fats and oils, are composed of carbon, hydrogen and oxygen. Chemically they are the glycerids of certain fatty acids, and may be readily split up into glycerin, stearic, palmitic and oleic acids. These acids, when free, unite with bases, sodium, potassium, calcium, etc., to form soaps. In the body hydrocarbons are normally either deposited in storage depots as a reserve, or completely split up into CO_2 and H_2O, and, as such, eliminated through the lungs. Their fuel value is very high, each gram (15 grs.) yielding 9 calories. If present in sufficient amount they are utilized for the production of heat and energy in preference to protein, and hence are described as "protein sparing." Carbohydrates, however, are still more readily oxidized, and are even better adapted to preserve the protein of the food and tissues from unnecessary combustion.

Under abnormal conditions, as in diabetes or starvation, the fats may be imperfectly metabolized, and the blood becomes laden with betaoxybutyric acid, diacetic acid and acetone. If this vice of metabolism is unchecked, the alkalinity of the blood is diminished, and the condition known as acid intoxication is induced. In order to neutralize the acidity, ammonia split off from the nitrogenous compounds combines with the acids. Under these conditions there is, therefore, an excess of ammonia eliminated in the urine. Therapeutically, it is well recognized that the administration of carbohydrates, if they have previously been withheld, will often overcome this complication of diabetes. Sodium bicarbonate administered by the mouth or intravenously (3 to 5 per cent. solution in water, or in physiologic saline solution) is sometimes effective. That it is not uniformly so is good

* Folin's method.

evidence that acidemia presents a profound disturbance of metabolism, not a simple alteration in the reaction of the blood. (See also Diabetes.)

Carbohydrates. The carbohydrates, like the hydrocarbons, consist of carbon, hydrogen and oxygen, but the hydrogen and oxygen are present in the same proportions as they are found in water. They are ingested in the form of insoluble starches (polysaccharids) or soluble sugars (monosaccharids and disaccharids). Prior to absorption they are converted into the monosaccharid glucose. Their caloric values vary slightly, but for clinical purposes the factor 4 is sufficiently accurate. In the tissues the glucose is split up or oxidized into CO_2 and H_2O, as in the case of the hydrocarbons, or is stored in the liver and muscles as animal starch, or glycogen. The glycogen serves as a reserve, and is always immediately available for the production of energy. Hydrocarbons and carbohydrates are best suited to furnish the heat and energy required by hard-working men. The old, and to some extent still the popular view, was that such individuals required an unusual amount of meat and other proteins. In tropic and temperate climates carbohydrates form the preferable source of energy. In the Arctic regions fats are preferred, as they furnish a far greater proportion of heat than other foodstuffs, bulk for bulk. Under normal conditions little or no sugar escapes in the urine, although the blood usually contains approximately 0.1 per cent. of sugar. As long as starchy food alone is ingested, the normal bounds are not exceeded, but even in normal persons the ingestion of a large amount of sugar (100 to 200 Gms.) may increase the percentage in the blood to such a degree that glucose will appear in the urine (limit of toleration). The secreting epithelium of the kidney holds back the sugar as long as it does not exceed a certain threshold value (0.14%). Beyond that value it is freely excreted. In some diseases of the pituitary and thyroid (hypopituitarism and hypothyroidism), the sugar toleration is increased, while in diabetes it is diminished. (For theory see Diabetes.)

Mineral Salts. A large assortment of mineral salts is required for growth and reconstruction, but these do not usually have to be especially provided, as they are supplied

in excess in any well-balanced diet. Occasionally there may be a deficiency, as of iron when an adult, for example, is kept for a long time on a pure-milk diet. The same is true of infants if an exclusive milk diet be maintained much beyond the usual period of lactation. In other instances—for example, in rickets—mineral salts, calcium phosphate and carbonate may be present in abundance in the diet, but, through same fault of absorption or metabolism, are not retained. The most important salt, quantitatively at least, is sodium chlorid. Sodium in the form of the chlorid, carbonate, etc., is present in the blood and tissue-juices to the extent of 0.7 per cent. Most individuals partake of an excess of sodium chlorid, which is quickly eliminated in the urine. If the excretory power of the kidney be impaired, as in certain forms of nephritis (parenchymatous), the salt is retained, and with it sufficient water to keep it in physiologic solution. This is believed, therefore, to be one of the most important causes of edema, so that the excessive use of salt may be far from innocuous. The ordinary foodstuffs, with the addition of merely enough sodium chlorid to make them palatable, will supply as much of this substance as is required for physiologic needs. In addition to sodium chlorid, other salts of the alkalies and alkaline earths, phosphates, sulphates, carbonates, chlorids, etc., of sodium, potassium, ammonium, calcium and magnesium, are eliminated in greater or less quantities in the urine. The following table, based on Folin's analyses of thirty "normal" urines, shows the average elimination of nitrogen, and of various important salts on a normal diet.

Certain substances, some of which contain salts, and others which are dependent for their activity on essential oils, etc., are employed in the diet as condiments. Many of these are absorbed and eliminated through the kidneys without exerting any distinct effects on metabolism. They act principally as stimulants to the secretions of the stomach and intestines, and secondarily, in some instances, as gastrointestinal and urinary antiseptics.

Vitamins. In recent years peculiar bodies, probably of a nitrogenous nature, which have a profound influence on nutrition have been found in very minute amounts in certain food-

TABLE I.

AVERAGE URINARY ELIMINATION ON FIXED DIET. COMBINED TABLE, AFTER FOLIN.[5]

Volume of Urine.	Total Nitrogen. (N_2).	Urea N.	Ammonia N.	Creatinin N.	Uric Acid N.	Undetermined N.	Total Sulphur as SO_3.	Total Phosphate as P_2O_5.	Chlorin.	Acidity.	Average Wt.
Mils	Grams	Grams	Grams	Grams	Grams	Grams	Grams	Grams	Grams	Mils	Kilograms
1430	16.0	13.9	0.7	0.58	0.12	0.60	3.31	3.87	6.1	617	63.4
		87.5%	4.3%	3.6%	0.8%	3.75%					

CORRESPONDING CONSTITUENTS IN DIET. AVERAGE OF NUMEROUS ANALYSES.

(Diet 119 grams protein, 148 grams of fat, 225 grams carbohydrate.)

Volume of Urine.	Total Nitrogen. (N_2).	Urea N.	Ammonia N.	Creatinin N.	Uric Acid N.	Undetermined N.	Total Sulphur as SO_3.	Total Phosphate as P_2O_5.	Chlorin.	Acidity.	Average Wt.
Mils	Grams						Grams	Grams	Grams		
2900	18.95*						3.74	5.78	6.13		
Total Fluid	Total N.						Total SO_3.	Total P_2O_5.	Total Cl.		

* The difference between the nitrogen ingested and that found in the urine is largely eliminated in the feces.

stuffs.[6] They have been dubbed vitamins. Beriberi has long been attributed to an exclusive diet of rice, but only within recent years has it been known that this affection could be avoided, or even cured by the use of unpolished—that is, unmilled—rice. The process of milling, if carried too far, removes the brownish covering (pericarp) which contains the prophylactic substance. This substance may be extracted from the refuse, and administered with curative effect. Beriberi is, therefore, in part at least, a deprivation disease. In scurvy the lack of fresh food—meat, vegetables or fruit-juices—is responsible for a similar state of affairs. For more than a century scurvy has been banished from the British and other navies, by the addition of lime-juice to the old dietary of ship's biscuit, "salt horse," and pudding. Recent researches make it evident that pellagra is a similar "deprivation" disease, due to a too-exclusive diet of salted meats and cereals, particularly Indian corn.

Water, Oxygen, Etc. Water constitutes between 80 and 90 per cent. of the human body, and a liberal supply is essential for the greatest efficiency in digestion and metabolism. It is to be remembered that the body is not entirely dependent for its water-supply on fluids ingested as such. Most of the so-called solid foods contain a liberal percentage of water, often as much as 70 per cent.,* while during the course of destructive metabolism considerable additional amounts of water ("chemical") are split off from the solid constituents of the diet. Water is eliminated principally through the kidneys, skin, lungs and intestines. The kidneys and skin, with a uniform supply, act reciprocally, to a large extent, in accordance with the degree of external heat. Although a liberal supply of water is advisable, the body shows a remarkable adaptability in this respect, and eliminates and retains water according to the demands of the metabolism. In cardiovascular and renal disease, an excess of fluid may be positively injurious, in that it burdens an overtaxed heart or exceeds the excretory power of the kidney.

Oxygen is absorbed into the blood through the lungs, and CO_2 is eliminated, in turn, through the same organs. The

* 1, white bread 35.6 per cent.; 2, ribs of beef, 70.9 per cent.; 3, halibut steak, 74.4 per cent.; 4, asparagus (cooked), 91.6 per cent.

amount of CO_2 eliminated bears a definite mathematic relation to the amount of O_2 intake. If the amount of oxygen inspired and the amount of carbon dioxid expired are estimated for a definite period, and the result is stated as a fraction ($\frac{CO_2}{O_2}$), we obtain what is known as the respiratory quotient. The respiratory quotient varies with different foodstuffs, so that by testing the gaseous interchange an expert observer may tell what form of food has been ingested. The respiratory quotient for carbohydrates is 1, for fat and protein 0.7 and 0.8, respectively.

General Metabolism. General metabolism is best measured in heat units or calories. The kilogram calorie, which is the caloric unit used in medicine, is that quantity of heat which is necessary to raise the temperature of one liter of water one degree centigrade. The caloric values of foodstuffs may be determined directly by burning them in the presence of oxygen in an apparatus known as a bomb calorimeter. Their physiologic caloric value is computed from the theoretic value by making due allowances for lack of absorption, and in the case of protein for incomplete utilization. As stated previously, protein and carbohydrate each yield 4 available calories per gram (15 grs.), while hydrocarbons yield 9 calories (dry weight). The factors of Rübner, though more often used, are less convenient, and no more accurate.* Alcohol, which, in small quantities is oxidized in the body with the production of heat, yields 7 calories per gram (15 grs.). Alcohol is useful to supplement the dietary in diabetes, and in that condition has a protein-sparing power similar to that of carbohydrates.

The caloric demands of the animal or human body may be actually determined in specially devised calorimeters. The heat production, under conditions of absolute rest and starvation, is known as the basal heat production. In man this amounts roughly to 1 calorie per kilogram (2.2 lbs.) per hour, or 24 calories per kilogram daily. A more accurate method is to state the daily requirements in terms of square meters of radiating skin surface. Stated in this form 35

* These are: protein and carbohydrate, 4.1 calories; fat, 9.3 calories, per gram.

calories per hour would be required for each square meter of surface.[7] If sufficient food be now given exactly to meet these theoretic requirements, it will be found in practice to be deficient, since the digestion and metabolism of the food in itself demands a certain amount of energy, roughly 10 per cent. This will bring the total daily requirements to 26.4 calories per kilogram per day. Calculating for a man of 70 kilograms (156 lbs.), or 2 square meters of superficial surface, the basal heat requirement plus the specific dynamic factor would amount to 1850 calories. During the waking hours, even while in bed, a certain additional amount of energy is expended in external movements, and still more with each additional degree of activity. Sitting in a chair, with the ordinary accompanying movements, involves a supplemental consumption of 20 calories per hour for an individual of 70 kilograms. Walking on the level is said to require in addition 160 calories per hour.* Ordinarily it is sufficient to remember that an adult at rest in bed requires approximately 30 calories for each kilogram of body weight per day (14 per lb.); if he follows a sedentary life, or one which involves only moderate exertion, he requires 35 to 40 calories (16 to 18 per lb.); with more vigorous exercise 50, or in very arduous occupations even as much as 60 calories per kilogram (24 to 28 per lb.) Babies and young children require quantities of food which are seemingly altogether out of proportion to the demands of their elders. This may be explained, in part, by their relatively large radiating surface in proportion to weight, and by their greater physical activity. In addition, a small amount is accounted for by the requirements incident to growth. An infant at birth requires as much as 100 calories for each kilogram of body weight per day (50 per lb.); by the end of the first year approximately 80 calories suffice; throughout childhood the requirements are appreciably higher than in adult life. A knowledge of caloric values and caloric requirements is particularly desir-

* $(70+7)\times 8 = 616$ calories (sleeping hours).
$(70+7+20)\times 16 = 1552$ calories (waking hours).
Two hours walk on level 320 calories.

2488 calories (in round numbers 2500).
(Taylor, A.: Digestion and Metabolism.)

Table II.

Liquid and Soft Diets.*

Description of Articles.	Description of Portions.						
	Rough measure.	Exact measure.		Protein.	Fat	Carbo-Hydrate.	Calories.
		Gms.	Oz. Av.	Gms.	Gms.	Gms.	
Milk, whole (4% fat)	A glassful or 7 fluidounces	217	7.6	7.1	8.7	10.8	150
Milk, skimmed (0.3% fat)	A glassful or 6⅔ fluidounces	205	7.2	7.0	0.6	10.4	75
Buttermilk (0.5% fat)	A glassful or 6¾ fluidounces	210	7.4	6.3	1.1	10.0	75
Whey (0.3% fat)	A glassful ("scant") or 6 fluidounces	187	6.6	1.9	0.6	9.4	50
Cream (18.5% fat)	1½ tablespoonfuls or ⅝ fluidounces	26	0.9	0.6	4.8	1.1	50
Butter (85% fat)	1 teaspoonful (rounded)	7	0.2	0.1	5.5	0.0	50
Sugar (granulated cane—powdered milk, malt, etc.)	2 teaspoonfuls (rounded) of granulated, 1 tablespoonful (heaping) of powdered	13	0.4	0.0	0.0	12.5	50
Oat or barley gruel (1 oz. of the flour to quart of water)	A glassful or 7 fluidounces	218	7.6	1.0	0.0	5.2	25
Legume gruel (1 oz. of the flour to quart of water)	A glassful or 7 fluidounces	217	7.6	1.7	0.0	4.6	25
Soda crackers (for use with milk)	1 cracker	6	0.2	0.6	0.5	4.4	25
Toast dried in oven (for use with milk)	1 thick (3″ x 3″ x ½″) or 2 thin slices, (wt. as bread: 39 Gms.)	23	0.8	3.6	0.5	20.4	100
Egg	One average (weight includes shell)	57	2.0	6.8	5.3	0.0	75
White of egg (in glass of water, lemonade or beef-extract)	Two average "whites"	49	1.7	6.0	0.1	0.0	25
Beef-juice (pressed—Holt)	2 tablespoonfuls	40	1.4	2.0	0.2	0.0	10
Beef-broth (Holt, mutton and chicken similar)	Large cup or 8 fluidounces	250	8.8	2.5	tr.	0.0	10
Olive oil (or cottonseed oil)	1 tablespoonful (even) ⅔ fluidounces	11	0.4	0.0	11.1	0.0	100
Gelatin (1 pckge. gelatin, ½ lb. sugar, 2 qts. water)	7 tablespoonfuls 3½ fluidounces	100	3.5	1.8	0.0	11.0	50
Junket (1 qt. milk, 2 oz. sugar)	8 tablespoonfuls 4 fluidounces	110	3.9	3.6	4.4	11.7	100
Soft custard (same with 4 eggs)	5 tablespoonfuls 2½ fluidounces	83	2.9	5.0	5.1	8.8	100
Cornstarch (1 qt. milk, 2 oz. sugar, 1 oz. of cornstarch)	7 tablespoonfuls 3½ fluidounces	100	3.5	3.3	4.0	13.0	100
Rice-pudding (1 qt. milk, 2 oz. sugar, 3 oz. of rice)	6 tablespoonfuls 3 fluidounces	83	2.9	3.3	3.3	14.4	100
Beef (round) scraped	2 small cakes	64	2.3	13.6	5.0	0.0	100
Chicken—young	1 slice	92	3.3	19.8	2.3	0.0	100

* Arranged for the Presbyterian Hospital, Philadelphia.

able in the treatment of obesity, malnutrition from any cause and diabetes. It is also useful in the dietetic treatment of many diseases outside of the group with which this section deals, for example, ulcer of the stomach and typhoid fever. Most of the dietetic tables found in current textbooks are derived directly or indirectly from the analyses of Atwater and Bryant.[8] The fuel values in their tables are calculated per pound, a method which is convenient for wholesale purchasing, but not for dietetic purposes. Locke, Fisher, and others[9] have variously modified these tables for practical purposes. Locke classifies the foods, and gives the caloric values and the chemical compositions of portions such as are ordinarily consumed. Fisher suggests an arbitrary unit of 100 calories, and gives the weight and composition of various foods which would furnish this amount of energy. In most cases these portions are not far from those usually taken; moreover, the convenience of this plan is not materially lessened if we employ in some cases fractional portions instead of whole units. The appended chart is based on this principle (Table II). Another convenient plan is to use simple units of 100 grams each. Taking the percentage figures for protein, fat and carbohydrate as given by Atwater and Bryant, one can readily obtain the caloric values desired by multiplying the figures for protein and carbohydrate by 4, and those for fat by 9, and adding the results together.

Table III has been calculated on this plan. In many instances simple fractions or multiples of 100 grams may be employed; 100 grams correspond roughly to 3½ ounces avoirdupois, or, in the case of liquids, to a little over 3 fluid-ounces (Apothecaries' measure).

TABLE III.

Soups.	P.	F.	C-H.	Cal.*
Bean-soup	3.2	1.4	9.4	63.0
Beef-juice	4.9	0.6		25.0
Beef-soup	4.4	0.4	1.1	25.6
Bouillon	2.2	0.1	0.2	10.5
Chicken-soup	3.6	0.1	1.5	21.3
Clam chowder	1.8	0.8	6.7	41.2
Consommé	2.5		0.4	11.6
Cream of asparagus	2.5	3.2	5.5	60.8

* P. = protein; F. = fat; C-H. = carbohydrate; Cal. = calories.

TABLE III—CONTINUED.

Soups—continued.	P.	F.	C-H.	Cal.
Cream of celery	2.1	2.8	5.0	53.6
Mock-turtle soup	5.2	0.9	2.8	40.1
Pea-soup	3.6	0.7	7.6	51.1
Tomato-soup	1.7	0.9	5.3	36.1
Meats.				
Porterhouse steak	21.9	20.4		271.2
Ribs of beef				
Lean	19.6	12.0		186.4
Fat	15.0	35.6		380.4
Round of beef				
Lean	21.3	7.9		156.3
Fat	17.5	16.1		214.9
Sirloin steak	18.9	18.5		242.1
Tenderloin broiled	19.8	11.8		185.4
Veal cutlet	20.3	7.7		150.5
Veal loin				
Medium fat	19.9	10.8		176.8
Lamb-chops, broiled	21.7	29.9		355.9
Lamb, leg (roast)	19.7	12.7		193.1
Mutton, leg	19.8	12.4		190.8
Pork, loin chops				
Medium fat	16.6	30.1		337.3
Pork-ribs	17.3	13.1		187.1
Ham, lean	25.0	14.4		229.6
Chicken—broilers	21.5	2.5		108.5
Fowl	19.3	16.3		223.9
Goose	16.3	36.2		391.0
Turkey, roast	27.8	18.4		276.8
Fish.				
Clams, round	6.5	0.4		29.6
Cod, fresh	11.1	0.2		46.2
Crabs, hard-shell	16.6	2.0		84.4
Haddock	17.2	0.3		71.5
Halibut steak	18.6	5.2		121.2
Lobster	16.4	1.8		81.8
Mackerel, fresh	18.7	7.1		138.7
Oysters, raw	6.2	1.2		35.6
Salmon, canned	21.8	12.1		196.1
Sardines, canned	23.0	19.7		269.3
Sea-bass	19.8	0.5		83.7
Eggs.				
Hen's eggs†				
Uncooked	14.8	10.5		153.7
Boiled	14.0	12.0		164.0
Vegetables.				
Asparagus cooked	2.1	3.3	2.2	46.9
Beans baked—canned	6.9	2.5	19.6	128.5
Beans, lima	7.1	0.7	22.0	122.7
Beans, lima—canned	4.0	0.3	14.6	77.1
Beans, red kidney—canned	7.0	0.2	18.5	103.8
Beans, string—cooked	0.8	1.1	1.9	20.7

† Two eggs weigh approximately 100 grams.

TABLE III—CONTINUED.

Vegetables—continued.	P.	F.	C-H.	Cal.
Beets, cooked	2.3	0.1	7.4	39.7
Cabbage	1.6	0.3	5.6	31.5
Carrots	1.1	0.4	9.3	45.2
Cauliflower	2.0	0.8	6.0	39.2
Celery	1.1	0.1	3.3	18.5
Corn, green	3.1	1.1	19.7	101.1
Corn, green—canned	2.8	1.2	19.0	98.0
Cucumbers	0.8	0.2	3.1	17.4
Olives	1.1	27.6	11.6	299.2
Onions, cooked	1.2	1.8	4.9	40.6
Parsnips	1.6	0.5	13.5	64.9
Peas, dried	24.6	1.0	62.0	355.4
Peas, green—cooked	6.7	3.4	14.6	115.8
Potatoes, boiled	2.5	0.1	20.9	94.5
Potato chips	6.8	39.8	46.7	572.2
Potatoes, mashed and creamed	2.6	3.0	17.8	108.6
Spinach, cooked	2.1	4.1	2.6	55.7
Sweet potatoes, cooked	3.0	2.1	42.1	199.3
Tomatoes, canned	1.2	0.2	4.0	22.6
Tomatoes, fresh	0.9	0.4	3.9	22.8
Breads and Cereals.				
Bread, home-made	9.1	1.6	53.3	264.0
Bread, rye	9.0	0.6	53.2	254.2
Bread, toasted	11.5	1.6	61.2	305.2
Bread, white—baker's	10.6	1.2	48.3	246.4
Crackers, Boston	11.0	8.5	71.1	404.9
Crackers, graham	10.0	9.4	73.8	419.8
Crackers, soda	9.8	9.1	73.1	413.5
Hominy, cooked	2.2	0.2	17.8	81.8
Macaroni, cooked	3.0	1.5	15.8	88.7
Oatmeal, boiled	2.8	0.5	11.5	61.7
Rice, boiled	2.8	0.1	24.4	109.7
Rolls, French	8.5	2.5	55.7	279.3
Shredded wheat	10.5	1.4	77.9	366.2
Dairy Products.				
Butter	1.0	85.0		769.0
Cheese, American pale	28.8	35.9		438.3
Cheese, cottage (no cream added)	20.9	1.0		92.6
Cheese, full cream	25.9	33.7		406.9
Cheese, Neufchatel	18.7	27.4		321.4
Cream	2.5	18.5		176.5
Evaporated cream	9.6	9.3		122.1
Skimmed milk	3.4	0.3		16.3
Fruits and Desserts.				
Apples	0.4	0.5	14.2	62.9
Bananas	1.3	0.6	22.0	98.6
Dates	2.1	2.8	78.4	347.2
Figs, dried	4.3	0.3	74.2	316.7
Grapes	1.0	1.2	14.4	72.4
Oranges	0.8	0.2	11.6	51.4
Orange marmalade	0.6	0.1	84.5	341.3
Peaches	0.7	0.1	9.4	41.3
Raisins	2.6	3.3	76.1	344.5
Strawberries	1.0	1.6	7.4	48.0

TABLE III—CONTINUED.

Fruits and Desserts—continued.	P.	F.	C-H.	Cal.
Almonds	11.5	30.2	9.5	355.8
Chestnuts	6.2	5.4	42.1	241.8
Peanuts	25.8	38.6	24.4	548.2
Pecans	9.6	70.5	15.3	734.1
Calf's foot jelly	4.3		17.4	86.8
Cup cake	5.9	9.0	68.5	378.6
Sponge cake	6.3	10.7	65.9	385.1
Pie, apple	3.1	9.8	42.8	271.8
Pudding, tapioca	3.3	3.2	28.2	154.8
Granulated sugar			100.0	400.0
Honey	0.4		81.2	326.4
Maple syrup			71.4	285.6

SCURVY (Scorbutus)

SCURVY IN ADULTS.[10]

Scurvy is a disease which is almost unknown in private practice, although well-marked cases are not at all uncommon in public institutions, and may be readily diagnosed if one bears the disease in mind. There have been many examples at the Philadelphia General Hospital, from which institution cases have been reported by Henry, Riesman, Jump and others.[11] Formerly a scourge of the sea, it is now more common on land. Aside from its sporadic occurrence, it appears in epidemic form in times of war, famine and pestilence. The "terrible malady of hunger" in Poland, recently (1916) described in the public prints, appears to have been scurvy, or something closely allied to it.

In the prodromal stages scurvy is characterized by mental apathy and depression, by extreme lassitude and muscular weakness, and by pallor, dyspnea and slight edema. In its fully developed form the foregoing symptoms are present, and, in addition, the gums are red and swollen, the lower extremities are dotted with petechiæ, and occasionally with larger hemorrhagic spots, and the subcutaneous tissues, in certain localities, are discolored by ecchymoses. There may also be subperiosteal hemorrhages. In the later stages there may be increasing anemia, cardiac weakness, dyspnea and edema, and death may occur from syncope. The usual complications are ulceration of the skin and of the mucous membranes, hemorrhagic effusion into the pleural, pericardial or

joint cavities, pulmonary gangrene, and bloody diarrhea.* The severer types of the disease are now excessively rare, dangerous complications are uncommon, and a fatal outcome is most unusual. Many theories have been advanced to account for the disease, notably that of Garrod (1848)[12] who attributed it to a lack of potassium salts; of Wright,[13] who considered it a manifestation of acid intoxication, and of Torup,[14] who incriminated ptomaine poisoning (from badly preserved or tainted meat and fish). Other writers[15] have thought it to be due to a specific infection seated in the gums or elsewhere. At present the trend of opinion is to group scurvy with the "deficiency diseases," such as beriberi, scurvy, pellagra and rickets. Casimir Funk[16] has designated this group by the general name of "Avitaminosen." He and others have attempted to isolate, by chemical means and animal experimentation, vitamins similar to those found curative in beriberi, but as yet this attempt has not been successful. Nevertheless, the vitamin theory seems to be the most reasonable of any yet advanced. Practically speaking, the etiology of scurvy is well known. It is brought about by a monotonous diet, consisting largely of salted or otherwise preserved meats and cereal foods, and deficient or entirely lacking in fresh fruits, green vegetables and tubers. A patient who was treated at the Philadelphia Hospital (service of Dr. Riesman) had subsisted on a diet consisting solely of sausage, bread and doughnuts. Canned fruits and vegetables may, or may not, possess antiscorbutic properties. These properties seem to be better preserved in the presence of organic acids, as in lime-juice, lemons, oranges and sauerkraut. Other contributory causes are bad air, filthy surroundings, insufficient clothing, exposure to wet and cold, hard work, and chronic diseases. All these conditions were to be found in the forecastles of the old-time sailing-ships; with the introduction of steam, voyages became short, and fresh food always available. Steam is thus given almost equal credit with lime-juice as a factor in the abolition of sea-scurvy.

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* In the polar regions "night blindness" is a common complication. The intense light of the arctic day exhausts the retina; scurvy is merely a predisposing cause.

There are very few *pathologic changes* typical of scurvy in adults. If incisions are made into the ecchymotic swellings in the neighborhood of the ankle, in the popliteal space and elsewhere, the subcutaneous tissues and muscles will be found to be infiltrated with bloody serum, or there may be dense clots of blood. The serous cavities may contain blood-stained effusions. The heart-muscle is usually soft and degenerated, and the spleen congested and softened. The blood-vessels have always been found normal. Complicated cases may show dysenteric ulceration and gangrene of the lungs.

The onset of the disease is gradual, and the patient may come to the hospital on account of extreme lassitude and inability to work. He is usually pallid ("earthy pallor"), and may be somewhat dyspneic. By the time he comes under observation the gums are swollen and of a deep-red color, but if the teeth are lacking this symptom may be absent. The skin is rough and dry, and minute hemorrhagic points are scattered profusely over the lower extremities. There may also be tender nodes along the tibia. In the neighborhood of the ankle, at the back of the knee, and in the thigh the loose subcutaneous tissues may be infiltrated, producing a superficial appearance of severe trauma: heat, redness, swelling and ecchymosis. These lesions of the gums, skin and subcutaneous tissues are considered by some authorities to be accidental complications[17] rather than essential symptoms of the disease. However, even if they are directly due to friction or slight trauma, they are indirectly referable to the state of the blood. Scurvy as we now see it is a benign disease with practically no mortality. The patients may appear the picture of misery, but a suitable diet will restore them to normal health within a few weeks at the most. The severer types of the disease are now almost unknown, but it would not be surprising if they should reappear in certain regions before the present European war is ended.

TREATMENT.

The prophylactic treatment of scurvy has for generations been prescribed by law in the navy and mercantile marine of almost all nations. The use of lime-juice was first introduced in the British navy, so that vessels of that nation were satiric-

ally described as "lime-juicers." The dietaries of hospitals, asylums, prisons and convict camps have, however, not always been above reproach; so that "deficiency diseases"—whether scurvy, pellagra or beriberi—have cropped out from time to time. Vedder[18] in a recent article has formulated some simple dietary rules which will suffice for the prevention of deficiency diseases:

1. "In any institution where bread is the staple article of diet, it should be made from whole-wheat flour.

2. "When rice is used in any quantity, the brown undermilled, or so-called hygienic rice, should be furnished.

3. "Beans, peas or other legumes, known to prevent beriberi, should be served at least once a week. Canned beans or peas should not be used.

4. "Some fresh vegetable or fruit should be issued at least once a week, and preferably at least twice a week.

5. "Barley, a known preventive of beriberi, should be used in all soups.

6. "If cornmeal is the staple of diet, it should be yellow meal or water-ground meal, that is, made from the whole grain.

7. "White potatoes and fresh meat, known preventives of beriberi and scurvy, should be served at least once a week, and preferably once daily.

8. "The too exclusive use of canned goods must be carefully avoided."

DIETARY.

In the treatment of a developed case of scurvy the diet will be the determining factor. This should include primarily orange-juice or lemonade, fruits, green vegetables—such as lettuce, cabbage and spinach—and potatoes. Secondarily, it should include an adequate supply of nourishing food, milk, eggs, fresh meat and bread, preferably whole-wheat bread. All these articles are more or less antiscorbutic, though less so than the fruit juices and the vegetables first mentioned. Fresh meat, containing the tissue juice and blood, is a good preventive, but preserved meats in which the animal juices have been replaced by preserving fluids are eminently conducive to this disease.[19] In the same way boiling and pas-

teurization detract from the antiscorbutic value of milk. Some, but not all, vegetables and fruits preserve their antiscorbutic virtue when canned or dried. This is particularly true of lime-juice, and various preparations of cabbage.

HYGIENE.

Faulty hygiene and unfavorable climatic conditions have played a large part in the history of scurvy. Like typhus fever, it is a disease which is associated in our minds with misery and squalor. It will be greatly ameliorated by good ventilation, light, cleanliness,—in fact, all those conditions which characterize a well-conducted hospital. It is quite unnecessary to specify these obvious matters in detail.

DRUGS.

At the first visit the patient will require a laxative, as the bowels are usually constipated, for which purpose Rochelle salts are best suited, as they act both as a laxative and antacid. Wright recommended that this drug be repeated several times a day, with a view of overcoming the acidosis which he postulated. He also recommended calcium chlorid to increase the coagulability of the blood. At the present day calcium lactate is to be preferred to the chlorid, 15 grains (1 Gm.) four times a day being a suitable dose.

For the loss of appetite any of the ordinary bitters may be prescribed. The secondary anemia would suggest the use of iron in some eligible form,—for example, as Blaud's mass. If there be a tendency to hemorrhage from the mucous membranes, an astringent preparation of iron would be preferable, such as the tincture of the chlorid.

The mouth should be carefully cleansed with potassium permanganate solution (0.1 per cent.), or a mild antiseptic solution such as liquor antisepticus (N. F.), and the gums touched with iodin. In case ulceration occurs, nitrate of silver (10 per cent.) may be applied locally. Emetin has been used without very definite results. Although all the suggestions which have been made have a rational basis, they are quite secondary to the diet, which, in the writer's experience, is in itself quite sufficient to effect a speedy cure in the ordinary case.

"Ship beriberi," which, according to Funk[20] was exhaustively investigated by Nocht in 1903, is closely related to scurvy in its etiology and symptoms. Funk says that the characteristic symptoms are anesthesia in the extremities, shortness of breath, and finally death from heart-weakness. In contrast to tropic beriberi, patients recover very quickly when they receive fresh provender.

INFANTILE SCURVY.*
(Barlow's Disease; Acute Rickets.)

Infantile scurvy is a constitutional disease due to unsuitable food, given for a prolonged period of time. It is an entirely preventable nutritional disorder. Although occasionally it develops among breast-fed infants, it is essentially a disease of the bottle-fed, and is common among those babies who are fed for some time upon proprietary food, condensed milk, sterilized, and boiled or pasteurized milk. Even buttermilk, prepared by heating, is a potential cause of infantile scurvy. Very rarely, a greatly diluted raw milk may produce scurvy, if the baby be kept upon it for a long time.

There must exist an "individual" predisposition to infantile scurvy,[21] since only a small proportion of improperly fed infants develop this disease.

Holt and Howland[22] note that only one of twins fed in exactly the same way developed scurvy. The so-called "exudative diathesis" of Czerny belongs definitely among the predisposing causes of infantile scurvy, which is the same disease as ordinary scurvy, but modified by the different conditions incident to infancy. Some chemical change, with destruction of the "vitamins," occurs in food prepared by overheating, which makes such food less assimilable. Infants fed exclusively upon such food show disturbances of metabolism, and infantile scurvy develops very gradually.

Infantile scurvy is associated with symptoms of rickets in about one-half of all cases, but there is no relation between the presence of the rickets and the development of the scurvy. While rickets results from an inability properly to digest improper food, which contains a deficiency of fat and an

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excess of carbohydrates, infantile scurvy is the direct result of unsuitable food, which is usually well digested.

Though scurvy has been noted in infants less than 1 month old, it is most frequently found in babies of between 6 and 15 months of age, who have been well previously. While rickets is a disease of the poor, scurvy more frequently occurs among the well-to-do. Surroundings play no rôle in the development of infantile scurvy, nor is there any history of previous illness; not even of frequent digestive disturbances.

There is undoubtedly an intimate causal association between scurvy, rickets and beriberi, since all three are primarily the result of improperly handled or prepared foodstuffs, in which the "vitamins" are either lacking or have been destroyed. The constituents of these foods have been so altered that the proper balance of the mineral salts within the organism is upset. While there is a definite loss of calcium in rickets, there is in scurvy an unexplained calcium retention.

Infantile scurvy, as described originally by Sir Thomas Barlow,[23] is characterized by (1) immobility, a pseudoparalysis, of the lower limbs especially, accompanied by excessive tenderness and by swelling, due mainly to subperiosteal hemorrhage, to edema with skin tense and shiny, and to thickening of the long bones, which is noted only after the swelling has disappeared; (2) swelling of the gums, generally with ecchymosis; (3) a tendency to hemorrhage; and (4) prompt recovery upon antiscorbutic diet. These symptoms develop gradually, with prolonged malnutrition, marked pallor, anemia and emaciation. The baby cries not only when handled, but even when approached; the gums become red and swollen, and bleed easily. If untreated, the tenderness spreads to practically all bones, with ecchymoses and hemorrhages in various parts of the body. Hess[24] noted enlargement of the right ventricle (as in beriberi), and a diminution of the clotting power of the blood.[25] Petechial hemorrhages into the skin and mucous membranes have been noted very early in the disease.

When not treated infantile scurvy is invariably fatal; moreover, these infants are most susceptible to any intercurrent infection, which may rapidly prove fatal.

Subperiosteal hemorrhages are constantly found near the epiphyseal junction, sometimes with separation of the epiphysis. This hemorrhage may extend into the muscles, though rarely into the joint. These hemorrhages are probably due to an existing hemorrhagic diathesis. They extend into the subcutaneous tissue, with exophthalmos in the orbit, or causing hematoma in the cheek or eyelid; into the gums, mucous or serous membranes, and even into the dura, kidney and spleen.

Microscopically, a rarefying process is noted in the long bones, with hemorrhages in the marrow and under the periosteum. The number of osteoblasts is decreased, and new bone is not formed. Bone already formed is well calcified, and absorption of bone is not increased.

In skiagrams a definite "line" is found at the junction of diaphysis and epiphysis in the long bones. This is noticed very early in infantile scurvy, even before cardinal symptoms appear. It persists for months, even after the infant apparently has recovered clinically. It is of value, especially in the diagnosis of the severe type of scurvy, with high fever and leucocytosis, to exclude acute pyogenic infection.

Infantile scurvy ought never to be mistaken for any other condition, since history and clinical findings are usually typical. Rheumatism does not occur in infancy; anterior poliomyelitis is readily distinguished by loss of the reflexes and examination of the cerebrospinal fluid. The skiagram also confirms the diagnosis. Traumatism, joint disease, spinal caries, and malignant bone disease may occasionally somewhat resemble scurvy.

While the undernourished infant may continue to increase in weight during the development of infantile scurvy, most babies fail to grow, either in weight or in length, but remain stationary during the development of scurvy. In infantile atrophy, on the contrary, growth in length is not affected, the infant failing to gain in weight alone.

TREATMENT.

It is very rare for a breast-fed baby to develop infantile scurvy; when this occurs, examination of the mother's milk will show its abnormal composition. Next to breast milk in

excellency comes raw cows' milk (certified milk), not sterilized or pasteurized. As certified milk is expensive, many babies must take pasteurized milk; in such instances orange-juice should also be given as a prophylactic, without regard to the age of the baby. If for any reason condensed milk or any proprietary food should be chosen, temporarily, clean raw milk should be used with it, and orange-juice given regularly also.

When the disease has already developed, all unsuitable food should be stopped at once. Here, again, raw cows' milk should be given. Spring-water is always advisable for these infants, ½ to 1 ounce (15 to 30 mils), warmed, about one hour before each feeding. Infants over 1 year old may also be fed any cereal containing the outer layers of the grain.

Because of the extreme tenderness, the infant should be kept at rest, undisturbed for several days, until the effect of the orange-juice is noted. In any severe case the affected limb may be kept in a splint for some days.

Orange-juice, the specific treatment for infantile scurvy, is given, ½ to 1 ounce (15 to 30 mils), every two or three hours, alone or diluted with spring-water. The juice of a whole orange should be given daily. Orange-peel may also be squeezed and added to the orange-juice. Lemon-juice is not so good, because it must be sweetened as well as diluted. Fresh grape-juice is of value when available.

For children over 1 year of age a mashed-potato gruel may also be given. Fresh green vegetables, such as spinach, well cooked, are also of value.

On account of the secondary anemia, syrup of the iodid of iron, 5 to 15 drops (0.30 to 0.92 mils), may be given three or four times a day. Beef-juice and codliver oil, so frequently forced upon infants with scurvy, are of no value at all, either in the prophylaxis or in the treatment of infantile scurvy.

RICKETS (Rachitis).*

Rickets is a chronic nutritional disorder of infants, characterized especially by the abnormal development of bone and cartilage, with subsequent deformity; the affection is trace-

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able directly to the prolonged feeding of indigestible food, and to faulty hygiene. Digestive disturbances constantly precede the development of rickets, which is attended by disturbances of metabolism and a definite loss of calcium salts.

Rickets has at least two definite causes, improper food and defective hygiene, especially lack of fresh air with defective air-space, due to confinement indoors. Therefore, it most commonly develops in winter. It usually affects infants 6 to 18 months old, and is slightly more frequent in boys than in girls. These babies are either bottle-fed or receive table food; in either case, improper food for the individual infant.

The importance of overfeeding in the causation of rickets has been discussed by Esser,[26] and recently by Eric Pritchard,[27] who believes that rickets invariably results from a relative excess of food, with the production of an acidosis. As the organism attempts to overcome this by compensatory overactivity of the hematogenetic centers in the long bones, the deformities of rickets develop.

The food given most of the infants who develop rickets has been found to contain little fat and a great excess of carbohydrates. The proprietary food, therefore, plays an important etiologic rôle. When rickets occurs among breast-fed babies, it will be found that either the mother is decidedly undernourished or has been nursing her baby over too long a period, or that the infant has also had table food. Rarely a child whose mother has previously nursed more than three children over a protracted period develops rickets, due to what Fordyce calls "previous lactational strain."[28]

Thus it follows that rickets is almost universal among the poor, especially in the large cities. While rare in the country, it also occurs there when an infant is improperly fed. It is most common among negroes in the United States, who get table food practically from birth, though they may also be breast-fed for two years or longer. It also occurs frequently in the breast-fed babies of foreign-born mothers whose breast milk shows the results of the mothers' malnutrition.

Although both parents and grandparents may have had rickets in infancy, this is readily explained by the fact that both the child's mother and grandmother may have been so undernourished that the milk secreted was also at fault.

Heredity, however, may exert some influence similar to that of those chronic diseases upon which Marfan[29] places such stress. He considers that rickets is always due to some chronic irritative process (alimentary, infectious, tuberculous or syphilitic). Syphilis is known to exist in many children who develop rickets, and very frequently alimentary disorders are present.

The normal growth of the bones is altered. The cartilaginous layer uniting the shaft and epiphysis is greatly widened and thickened. The transitional zone is softer than normal cartilage, blending with the epiphyseal cartilage on one side, but showing an irregular dentate border on the other. Fibrous tissue replaces normal red bone-marrow near the epiphysis. A softer and more vascular bone results, unnaturally flexible, readily producing deformities and fractures. Calcium is excreted in excess, so that the occurrence of much "limeless" bone is the most striking feature in rickets.

Definitely constant changes are noted in skiagrams: delayed ossification and indistinct epiphyses; fraying out of the end of the shaft next the epiphyseal line; broadening of the end of the shaft next to the epiphysis; cortical thickening on the concave side of the curved bones; and areas of diminished density in the bone shadow of the shaft. In the acute stage, besides, periosteal thickening and multiple fractures may be observed.

The development of rickets is gradual, and is always preceded by several attacks of digestive disturbances. Excessive perspiration about the head, restlessness, constipation, frequent coryza, bronchitis, pallor with secondary anemia, and a tendency to muscular spasm such as laryngismus stridulus, nystagmus, tetany or convulsions are noted. Dentition is delayed, and the anterior fontanelle remains widely open for two years or longer. Craniotabes, thickening of the bones of the forehead, enlarged epiphyses at wrists and ankles, "beading" at the costochondral junctions ("rosary"), "pigeon" or "chicken" breast; "funnel" chest, Harrison's groove, greenstick fractures, knock-knee, bowlegs, and kyphosis (probably postural), are also noted. The muscles become flabby and the ligaments are relaxed, the lymph-glands enlarge, as well

as the liver and spleen, and the abdomen becomes prominent ("pot-belly").

While rickets itself is never fatal, infants with rickets seem very prone to take other infections, and to die from them rapidly.

TREATMENT.

As properly fed babies do not develop rickets, every baby should be fed upon breast-milk, or upon a well-balanced cows' milk mixture, given in proper quantities at regular intervals. It is important that the pregnant woman and the nursing mother learn to eat enough nourishing food, and to drink sufficient water. Babies should not be weaned until 9 or 10 months old, nor should breast-feeding be continued longer than one year. When the mother's milk supply begins to fail, it should be supplemented by a bottle given regularly after both breasts every three or four hours.

For infants over 1 year of age a gradually increasing variety of other foods, with a gradual diminution in the amount of milk given, will prevent rickets. Such foods may be well-cooked cereals, strained at first; stale bread or toast with butter; fruit juices and stewed fruits; baked and mashed potatoes with butter; well-cooked green vegetables; soft-boiled eggs; and a little meat, well chopped up, two or three times a week. Beef-juice, meat-soups and meat-broths are valueless, and may be even harmful. Babies should neither be overfed nor underfed.

Regular bathing in lukewarm or cool water (80° to 90° F., 27° to 30° C.); much fresh air and sunlight; enough, and not too much, clothing, are all-important. Care should be exercised to prevent the occurrence of any illness, especially respiratory and digestive disturbances.

As all active rickets have ceased when the child reaches the age of 2 years, treatment will only be valuable earlier. Whatever trace of rickets remains after 2 years of age will, of itself, disappear as the child grows older, always excepting the severe deformities.

DIET.

When the breast-fed baby develops rickets, the mother must take enough nourishment and exercise out-of-doors; the

baby must be fed regularly, not too fast, not too much, and not too often. The breast-fed baby should receive no other food; nothing else except 4 or 5 teaspoonfuls of hot-water about one hour before nursing.

When the bottle-fed baby develops rickets, the formula must be changed, giving a certified milk mixture containing enough protein, a gradual increasing percentage of fat, and not too much sugar. In severe cases in very young infants, a wet-nurse is advisable. In the baby of 10 months or more, a rapid change to the varied diet, with less milk, is always best.

HYGIENE.

Babies with rickets need abundant fresh air and sunlight. For this nothing can approach the beneficial effect of the seashore, even in winter. A change of air alone, from city to seashore, or country, is always followed by rapid improvement.

The regular daily bath is best given in salt water, lukewarm or cool. In some cases cold sponging is of great value. In either case, the bath should be followed by rubbing the extremities and spine with olive oil or cocoa-butter. This affords the slight massage necessary to overcome the muscle relaxation. In a few, more advanced cases, passive movements, resistant exercises, and even electricity, are indicated. In older children, without deformity, exercises, even walking, are to be encouraged.

DRUGS.

The only drugs of any value in the treatment of rickets are codliver oil and phosphorus. These are best combined, using $\frac{1}{250}$ to $\frac{1}{100}$ grain (0.00026 to 0.00065 Gm.) of phosphorus to ½ to 1 dram (1.90 to 3.75 mils) of a 50 per cent. emulsion of pure codliver oil, given three times a day after meals. The phosphorus may be used alone in some cases. When there is marked anemia, iron may be added to these, either as the pyrophosphate (¼, ⅓ or ½ grain; 0.01620, 0.02160 or 0.03240 Gm.), or the syrup of the iodid (5 to 15 drops; 0.32 to 0.92 mil).

When the baby's appetite is poor, tincture of nux vomica (3 to 7 drops; 0.18 to 0.42 mil) will be of great value, preferably

in combination with an equal quantity of bicarbonate of soda and twice as much compound infusion of gentian, given three times a day before meals, with water.

Constipation is overcome by regulating the baby's milk formula, giving fruit juices and stewed fruit, and by using a soap suppository, since most of these infants simply seem unable to start defecation. When a laxative is needed besides, magnesium sulphate (½ to 1 teaspoonful [1.90 to 3.75 mils] in 2 or 3 teaspoonfuls [7.50 to 11.25 mils] of distilled water), or milk of magnesia (½ to 2 f3; 1.90 to 7.50 mils), will help.

Olive oil, lime-salts, even phosphates and organotherapy have all proved useless in the treatment of rickets.

When there is a marked tendency to muscle-spasm (spasmophilia), a hot bath, hot pack or mustard bath, with a laxative, may help. Rarely small doses of bromids are useful at bedtime.

As the kyphosis is primarily postural, keeping the baby on its back and not allowing it to sit up much will gradually bring about recovery. Splints, casts, or braces may be of value for the bow-legs or knock-knees later, but at first massage alone will accomplish much. Gymnastics are good to overcome chest deformities; and in later childhood (5 to 8 years of age) the severe bony deformities may require surgical intervention.

CONGENITAL RICKETS.

Holt and Howland[30] state that there is probably no such condition, although a congenital type has been described on the Continent, and attributed to malnutrition and to chronic disease in the mother. Investigations do not show that babies are born with rickets, but one may believe that a tendency or predisposition to the development of rickets exists as a congenital defect.[31]

LATE RICKETS.
(Rachitis Tarda; Adolescent Rickets).

This is also not found in the United States, but has been described abroad. It usually occurs in girls at the age of puberty, and affects the lower extremities and the spine espe-

cially, producing much pain and great deformity. A "rosary," enlargement of the epiphyses, scoliosis, asymmetric deformities of the legs, headache, and marked physical and intellectual torpor are noted. While the treatment is exclusively orthopedic, Marfan[32] claims results from adrenalin, using 15 to 20 drops of a 1 : 1000 solution three times daily.

OBESITY.

Obesity was not unknown even to the ancient Greeks, in spite of their love for grace of form and devotion to gymnastic exercise. Hippocrates (according to Immerman) for corpulence advised a scanty diet, cold baths, and exposure of the unclothed body to the open air. A similar mode of treatment is said to be consciously practised on the coast of Italy, but in this country the combination of exposure to the open air and bathing has not acquired any reputation as a reducing measure. The modern interest in obesity dates in a rough way from the time of Banting, who, in an open "Letter on Corpulency" (London, 1863), narrated how his physician (Dr. William Harvey) had cured him of this distressful malady by means of a diet as pleasant as it was effectual. This ingenuous epistle, while it aroused much merriment, created a tremendous vogue for reduction cures. The subsequent *régimes* of Ebstein, German Sée, Oertel, von Noorden, Moritz (Karell), and others, have been based in a continually increasing measure on the fundamental studies of nutrition, inaugurated by Voit and continued by Ruebner, Atwater, Chittenden, and a host of others. The admirable monograph of von Noorden[33] and the recent review of Matthes[34] contain complete bibliographies. The author is indebted to von Noorden's work for many of the facts embodied in this article.

Simple or alimentary obesity (Fettleibigkeit) may be defined as an undue accumulation of adipose tissue, due to an immoderate ingestion, relative or absolute, of carbonaceous foods (fats, sugars and starches). This fatty accumulation is more or less uniformly distributed throughout the subcutaneous connective tissues, in the mediastinum, mesentery, omentum, and in and about the principal organs. Von Noorden

divides this type of obesity, which he designates as "exogenous," into forms due to (1) overeating, (2) lack of exercise, or (3) to a combination of both factors. A large proportion of all cases of excessive corpulence fall under this caption, and are susceptible to simple measures: restriction of diet, exercise (climbing, gymnastics, sports), baths, etc. The diet must be reduced to a figure which will cause a slight excess of expenditure over income, or the output of heat and energy must be increased to a sufficient extent to attain the same end.

Closely allied to the cases of simple obesity are certain exceptional ones which do not yield readily, or at all, to dietetic restrictions, even in the hands of experts. This group is variously designated by clinicians as: endogenous, constitutional, glandular, arthritic, or toxi-infectious obesity (Fettsucht). It is held by some authors that there is a qualitative alteration in metabolism, due to an "arthritic diathesis," to anomalies of the internal secretions, or to toxins (*e.g.*, after typhoid fever). It is more satisfactory, however, to explain these cases by assuming an unusual economy in all the life processes, rather than any deviation from the ordinary principles of metabolism. Thus it is quite certain that the external movements incident to an ordinary quiet life, differ enormously in persons of varying temperaments under approximately identical conditions.*

A third division of obesity comprises certain well-defined syndromes, the so-called lipomatoses, which are characterized by a peculiar disposition of adipose tissue, disorders of the endocrine glands, and nervous symptoms. The most important of these lipomatoses are Dercum's disease (adiposis dolorosa) and Fröhlich's syndrome (degeneratio adiposo-genitalis).

It is a good clinical rule to treat the cases comprised under the first two headings (exogenous and endogenous) as exogenous obesity until therapeutic failure or distinct evidences of glandular insufficiency place them definitely in the second class. It is sufficiently evident that the deposition of fat in

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* This can be tested roughly by the use of a pedometer.

itself presents nothing abnormal, and only becomes distinctly pathologic when it interferes with the normal activity of the patient or seriously impairs the functions of important organs. In the milder cases personal whims, fashion, or custom fix the limits between embonpoint and undue corpulence. Americans in the past were generally credited with being a spare, wiry people—due primarily, no doubt, to the active life characteristic of a new country—and we still prefer the slender types. This is in marked contrast to some of the older countries, where a moderate degree of rotundity is evidently looked upon as desirable. The physician is sometimes called upon to urge a reduction "cure," or, at least, a restriction of diet in certain sleek, "contented" individuals, or, on the other hand, to discourage such measures in young women who are obsessed with the idea that they are growing fat. As an aid to the determination of the optimum weight, numerous tables have been prepared ostensibly furnishing the physician with the normal range of weight for any given age, sex, height, or even race (Teuton, Latin). Many of these are from life-insurance statistics, and are presumably computed from a class of persons more prosperous and better fed than the average citizen. At any rate, the figures usually given are excessive, if applied to persons of slender ("gracile") build and slight muscular development. Such persons would be quite unwieldy if they attained a "normal" weight. A relative obesity may also occur in persons crippled by disease (arthritis), or deformity (loss of a limb).

The following tables are used by the Penn Mutual Life Insurance Company of Philadelphia (courtesy of Dr. Harry Toulmin, Medical Director):

TABLE OF HEIGHTS AND WEIGHTS AT VARYING AGES.

(The bold-faced figures are 20 per cent. over and under the average).

MEN.

Ages	15-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69
Ft. In.										
5 0....	**96**	**100**	**102**	**105**	**106**	**107**	**107**	**107**	**105**	...
	120	125	128	131	133	134	134	134	131	...
	144	**150**	**154**	**157**	**160**	**161**	**161**	**161**	**157**	...
5 1....	**98**	**101**	**103**	**105**	**107**	**109**	**109**	**109**	**107**	...
	122	126	129	131	134	136	136	136	134	...
	146	**151**	**155**	**157**	**161**	**163**	**163**	**163**	**161**	...

TABLE OF HEIGHTS AND WEIGHTS AT VARYING AGES.
(The bold-faced figures are 20 per cent. over and under the average).

MEN.

Ages		15-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69
Ft.	In.										
5	2....	**99**	**102**	**105**	**106**	**109**	**110**	**110**	**110**	**110**	...
		124	128	131	133	136	138	138	138	137	...
		149	**154**	**157**	**160**	**163**	**166**	**166**	**166**	**164**	...
5	3....	**102**	**105**	**107**	**109**	**111**	**113**	**113**	**113**	**112**	**112**
		127	131	134	136	139	141	141	141	140	140
		152	**157**	**161**	**163**	**167**	**169**	**169**	**169**	**168**	**168**
5	4....	**105**	**108**	**110**	**112**	**114**	**115**	**116**	**116**	**115**	**114**
		131	135	138	140	143	144	145	145	144	143
		157	**162**	**166**	**168**	**172**	**173**	**174**	**174**	**173**	**172**
5	5....	**107**	**110**	**113**	**114**	**117**	**118**	**119**	**119**	**118**	**118**
		134	138	141	143	146	147	149	149	148	147
		161	**166**	**169**	**172**	**175**	**176**	**179**	**179**	**178**	**176**
5	6....	**110**	**114**	**116**	**118**	**120**	**121**	**122**	**122**	**122**	**121**
		138	142	145	147	150	151	153	153	153	151
		166	**170**	**174**	**176**	**180**	**181**	**184**	**184**	**184**	**181**
5	7....	**114**	**118**	**120**	**122**	**124**	**125**	**126**	**126**	**126**	**125**
		142	147	150	152	155	156	158	158	158	156
		170	**176**	**180**	**182**	**186**	**187**	**190**	**190**	**190**	**187**
5	8....	**117**	**121**	**123**	**126**	**128**	**129**	**130**	**130**	**130**	**130**
		146	151	154	157	160	161	163	163	163	162
		175	**181**	**185**	**188**	**192**	**193**	**196**	**196**	**196**	**194**
5	9....	**120**	**124**	**127**	**130**	**132**	**133**	**134**	**134**	**134**	**134**
		150	155	159	162	165	166	167	168	168	168
		180	**186**	**191**	**194**	**198**	**199**	**200**	**202**	**202**	**202**
5	10....	**123**	**127**	**131**	**134**	**136**	**137**	**138**	**138**	**139**	**139**
		154	159	164	167	170	171	172	173	174	174
		185	**191**	**197**	**200**	**204**	**205**	**206**	**208**	**209**	**209**
5	11....	**127**	**131**	**135**	**138**	**140**	**142**	**142**	**142**	**144**	**144**
		159	164	169	173	175	177	177	178	180	180
		191	**197**	**203**	**208**	**210**	**212**	**212**	**214**	**216**	**216**
6	0....	**132**	**136**	**140**	**143**	**144**	**146**	**146**	**146**	**148**	**148**
		165	170	175	179	180	183	182	183	185	185
		198	**204**	**210**	**215**	**216**	**220**	**218**	**220**	**222**	**222**
6	1....	**136**	**142**	**145**	**148**	**149**	**151**	**150**	**151**	**151**	**151**
		170	177	181	185	186	189	188	189	189	189
		204	**212**	**217**	**222**	**223**	**227**	**226**	**227**	**227**	**227**
6	2....	**141**	**147**	**150**	**154**	**155**	**157**	**155**	**155**	**154**	**154**
		176	184	188	192	194	195	194	194	192	192
		211	**221**	**226**	**230**	**233**	**235**	**233**	**233**	**230**	**230**
6	3....	**145**	**152**	**156**	**160**	**162**	**163**	**161**	**158**	...	...
		181	190	195	200	203	204	201	198	...	...
		217	**228**	**234**	**240**	**244**	**245**	**241**	**238**	...	...

TABLE OF HEIGHTS AND WEIGHTS AT VARYING AGES.
(The bold-faced figures are 20 per cent. over and under the average).

WOMEN.

Ages Ft.	In.	15	20	25	30	35	40	45	50	55
4	11..	**83**	**87**	**90**	**93**	**95**	**98**	**100**	**102**	**103**
		104	109	113	116	119	122	125	127	129
		125	**130**	**135**	**139**	**143**	**147**	**150**	**153**	**155**
5	0..	**85**	**89**	**92**	**95**	**97**	**100**	**102**	**104**	**106**
		106	111	115	118	121	125	128	130	132
		127	**133**	**138**	**142**	**146**	**150**	**153**	**156**	**158**
5	1..	**87**	**91**	**94**	**97**	**99**	**102**	**105**	**107**	**108**
		109	114	117	121	124	128	131	133	135
		131	**136**	**141**	**145**	**149**	**153**	**157**	**160**	**162**
5	2..	**89**	**93**	**96**	**99**	**102**	**104**	**107**	**109**	**111**
		112	117	120	124	127	131	134	136	139
		134	**140**	**144**	**148**	**153**	**157**	**160**	**163**	**166**
5	3..	**91**	**95**	**98**	**101**	**104**	**107**	**109**	**112**	**113**
		114	119	123	127	130	134	137	139	141
		137	**143**	**148**	**152**	**156**	**160**	**164**	**167**	**170**
5	4..	**94**	**98**	**101**	**104**	**107**	**110**	**112**	**114**	**116**
		117	122	126	130	133	137	140	143	145
		140	**147**	**151**	**156**	**160**	**164**	**168**	**171**	**174**
5	5..	**96**	**100**	**104**	**107**	**110**..	**113**	**116**	**118**	**119**
		120	126	130	133	137	141	144	147	149
		144	**151**	**156**	**160**	**164**	**169**	**173**	**176**	**179**
5	6..	**99**	**103**	**107**	**110**	**113**	**116**	**118**	**121**	**123**
		123	129	133	137	141	145	148	151	153
		148	**155**	**160**	**164**	**169**	**173**	**178**	**181**	**184**
5	7..	**102**	**106**	**110**	**113**	**116**	**118**	**122**	**124**	**126**
		127	133	137	141	145	148	152	155	158
		152	**159**	**164**	**169**	**174**	**177**	**183**	**186**	**190**
5	8..	**105**	**109**	**113**	**116**	**120**	**123**	**126**	**128**	**131**
		131	137	141	145	149	154	157	161	163
		157	**164**	**169**	**174**	**179**	**184**	**189**	**193**	**196**
5	9..	**108**	**113**	**116**	**120**	**123**	**127**	**130**	**133**	**136**
		135	141	146	150	154	158	162	166	169
		162	**169**	**175**	**180**	**185**	**190**	**195**	**199**	**202**

In the absence of tables the simple rule popularized by Moritz may be used to approximate the weight. There are more complex formulas, but as they all include variables, their advantages do not counterbalance their complexity. Moritz subtracts 100 from the height, expressed in centimeters,

and takes the balance as a measure of the weight in kilograms.*

The normal weight varies with age and sex; thus, at a certain stage of adolescence girls exceed boys in height and weight, while before and after the reverse is true. Children and adolescents are occasionally obese; this may be a manifestation of cretinism or myxedema, but is more frequently the result of dietetic errors. Young adults continue to increase in weight till a maximum is reached in middle age ("fat and forty"). In advanced age there is likely to be a slight decline. Women are more subject to obesity than men. Their indoor occupations entail less expenditure of energy than the outdoor activities of the other sex. During pregnancy and lactation, moreover, the deposition of fat is an important conservative function which is sometimes encouraged beyond the bounds of reason. With advanced years and the advent of the menopause, physiologic demands are much diminished, but the acquired surplus is likely to persist. The prevalence of obesity in women may be due, in part, to their fondness for sweets. The influence of heredity is very evident in certain families, or even in whole races (Jewish). Anders asserts that heredity was distinctly traceable in more than 60 per cent. of his cases of obesity. Von Noorden suggests that dietetic and culinary habits may be handed down from generation to generation—naturally on the female side—and of themselves suffice to account for the prevalence of unusual rotundity in certain families.

The chief hygienic factors concerned in the production of obesity may be comprised under the headings food, drink and physical activity. In certain occupations several, or all, of these factors are combined with maximal effect. The quality and quantity of the food must be considered. Protein has little or no influence on the deposition of fat, but carbohydrates, and to a less extent, fats are of dominating importance. Alcoholic beverages, particularly those rich in saccharin derivatives (sweet wines and malt liquors), are very conducive to

* If the height is known in inches divide by 0.3937 to convert into centimeters; if the weight be found in kilograms multiply by 2.2 to obtain pounds. For example a man of 67 inches in height or 170 centimeters should weigh 70 kilograms or 154 pounds.

an increase of weight.* Alcohol in itself has a high caloric value (7 calories per gram), and if consumed in moderate amounts may be completely utilized, thus sparing an appreciable amount of fat and carbohydrate. The effect of an excessive fluid intake is more questionable.

Simple obesity is not a disease, but merely an exaggeration of a normal state, and if uncomplicated is unaccompanied by symptoms, except those due to mechanical causes. Excessive weight interferes with walking and other movements, and induces slight dyspnea on account of the increased work thrown upon the heart and lungs. There is also a tendency to free perspiration. If the deposits are localized, about the heart, for example, there may be more serious disturbances of function. In persons of a gastroptotic habit, a localized deposit of fat in the mesentery and abdominal wall may be decidedly beneficial. The deposition of fat is not uniform, even in simple obesity, *i.e.*, certain localities are more likely to suffer than others. While the feet, hands, and face are often spared, the abdominal wall, and in women the buttocks, thighs and breasts are sites of predilection. In the endogenous forms of obesity there is a qualitative difference in the fatty infiltration which tends to invade the muscular fibers, cardiac or skeletal, and this may seriously compromise their function. Obesity tends to persist indefinitely, though it may occasionally disappear spontaneously in old age. While not in itself serious, it frequently predisposes to other diseases, so that obese subjects, on the average, are shorter-lived than those of sparer habits.

Subjects of obesity may be of phlegmatic temperament, but if this tendency be unduly marked it should suggest the possibility of thyroid insufficiency. Similarly neuralgia and hyperesthesia should make one think of Dercum's disease. Apoplexy is not an uncommon terminal event; it is to be attributed to concomitant vascular disease.

Anemia predisposes to fat accumulation, and there is a well-marked type of anemic obesity which is to be contrasted with the "plethoric" or "full-blooded" type. In such cases

* Banting considered the following articles particularly conducive to obesity: bread, butter, sugar, potatoes, milk, beer, port and champagne.

tonics are essential, and extreme reduction measures are inadvisable.

There is a close relation between obesity and disturbances of the glands of internal secretion. Certain other glands which primarily have an external secretion, the pancreas, testicles and ovaries, are capable of exerting similar effects. Hyperfunction of the thyroid tends to increase metabolism and to induce loss of weight, while hypofunction, as seen in an exaggerated degree in myxedema and cretinism, leads to an increased deposition of fat. Symptoms which should suggest a thyroid element in a given case are somnolence, alopecia, dryness of the skin, cold feet and hands, and diminished sweating. In hypopituitarism obesity is associated with maldevelopment of the sexual organs, scanty or atypical hair distribution, and large breasts (in the male). The influence of the sexual glands on fat distribution is manifested by the normal differences in the two sexes, as well as by those changes which result from castration. Conversely, lessened sexual desire, impotence, scanty menstruation, and sterility are credited to excessive obesity. In eunuchs there is a marked liability to an obesity which partakes of the female type. It is a question whether the well-recognized tendency to fleshiness in women who have been deprived of their ovaries by operation, or have reached the menopause, is to be attributed to the withdrawal of glandular influence or to extraneous causes.*

The relation between obesity and diseases of the heart is a close one. Simple obesity may lead to cardiac embarrassment and ultimately to hypertrophy and dilatation, on account of the excessive accumulation of fat in and about the heart. On the other hand, primary cardiovascular disease is often complicated by obesity. The best results from reduction cures, fluid limitation, hydrotherapy, graduated exercises, and the like, are observed in cases of obesity complicated by cardiac disease.

In diseases of the respiratory system overweight is often of serious import, on account of the burden thrown upon the

* A German author recently followed up his cases of oöphorectomy, and was unable to determine any special tendency to fat accumulation.

respiratory as well as the circulatory organs. It is a serious complicating factor in pneumonia, chronic bronchitis, emphysema, asthma, and even in pulmonary tuberculosis. In the last-named disease overfeeding may lead to an anemic type of obesity, which cannot be considered a valuable asset to the patient.

Obese subjects frequently possess an unusually good digestion and power of assimilation. This probably accounts for their ability to gain on a diet which is apparently not excessive. Disturbances of the digestive system are not common. The liver is occasionally enlarged as the result of fatty infiltration.

Renal complications include passive congestion of the kidneys and chronic diffuse nephritis. Both are usually associated with cardiovascular disease. Diseases of the muscles, bones and joints bear no direct relation to excessive corpulence, but, if disabling, may act as predisposing causes, by limiting motion and reducing the output of energy. Skin complications, chiefly intertrigo and eczema, are common on account of the apposition of fat-laden folds of skin, for example, beneath the breasts. After rapid weight reduction, unsightly "striæ" are observed upon the breasts, abdomen and thighs. Finally, obesity is frequently complicated by other metabolic disturbances, particularly gout and glycosuria. This is sufficiently accounted for by the similarity of the predisposing causes: overeating, overdrinking, lack of exercise, and similar dietetic defects.

French authors describe an arthritic diathesis which they believe predisposes to a large group of closely related diseases: obesity, gout, diabetes, nephritis, stone, migraine, asthma, neurasthenia, eczema, acne, purpura and urticaria. To most of us the connection between these varied conditions is not so obvious, although occasionally it is supported by therapeutic observations.

TREATMENT.

Prophylaxis. The preventive treatment of obesity is more important, and more effectual than the curative treatment. In childhood and old age it is usually the only safe method of attack. It is the duty of the physician to warn adult patients

who are tending to overweight of the possible injury to the circulatory or other systems, and after investigation of habits and diet to suggest appropriate measures of prevention. These may suffice to check further increment. In children the general rule is to avoid any severe measures of reduction, and particularly any restriction of protein food, as this may interfere with proper development. The child's diet should be studied from carefully written records kept by the mother or other observer. In many cases gross dietetic errors will thus be discovered, usually an excessive ingestion of sugars and starches. In children it is best to give the carbohydrates in the form of well-cooked starches, bread, macaroni, rice, breakfast cereals, and plain puddings, rather than in the form of cakes, sugars, candies and other sweets. In these cases, as in the treatment of obesity generally, the diet should be adjusted to the normal weight for the age and height, rather than to the actual weight. The allotment of protein should be ample to provide for the growing organism, and to guard against the robbing of the body protein, which is liable to occur with even slight restriction of carbonaceous foods. In adolescence mild reduction cures carried out under close surveillance are admissible.[35]

Exercise plays a more important rôle in children and adolescents than in adults, and in cases uncomplicated by cardiac weakness it should be encouraged to the fullest extent. Open-air sports, such as swimming, rowing, walking, running, bicycling, basket-ball, base-ball and tennis are far preferable to indoor gymnastics. Exercise develops the muscular system at the cost of the fatty tissues, and even if obesity persists, it is less serious in the well-muscled than in the flabby, anemic individual.

In a small proportion of obese children, careful scrutiny will elicit symptoms of hypothyroidism or dispituitarism. For the latter there is no satisfactory specific treatment. In hypothyroidism (cretinism) excellent results may be obtained by the use of *dried thyroids* given in small doses, ⅓ grain (0.2 Gm.) three times a day, and increased if well borne. If the diagnosis has been well grounded, remarkable results may be expected. In the aged, severe restriction of diet, active exercise and hydrotherapeutic measures are alike inadmissible.

The diet may be kept within normal bounds, and physical measures such as massage and passive movements employed.

THE TREATMENT OF UNCOMPLICATED OBESITY IN ADULTS.

General Principles. The caloric requirements of adults under varying conditions have been considered in the introductory chapter on Metabolism (*v. s.*). For men and women pursuing a life of moderate activity, but without excessive labor, approximately 35 calories per kilogram (corresponding to 16 calories per pound) are ordinarily sufficient. In obesity it is necessary to reduce the caloric intake with or without increasing the energy output. This should be done by reducing the carbonaceous foods (fats and carbohydrates). The protein should be maintained at its normal level (1 Gm. per Kg.), or even increased 50 or 100 per cent. A liberal protein ration is of practical advantage for several reasons—practical and theoretical. (1) It makes the necessary restriction of other foodstuffs endurable. (2) Proteins, even if increased beyond the usual amount, are not conducive to increase of weight. (3) A liberal provision of protein food is a protection to the vital tissues, sometimes impaired by too strict dieting. Thus, if fats, sugars and starches are limited and the protein ration is insufficient, carbohydrate may be split off from the body protein and consumed. The caloric value of the diet may be reduced by restricting either the carbohydrate or the fat indifferently, or, in most cases, by limiting both.

The famous regimens of Banting and Oertel, in accordance with the ideas of their day, gave a very liberal allowance of protein (150 to 170 Gms.), but restricted the fats and carbohydrates very closely. They resemble a strict diabetic diet without fat. Such a diet can easily be constructed by the aid of the tables given under "Diabetes." Ebstein's diet was less rich in protein (100 Gms.), but permitted the use of a fairly large amount of fat (85 Gms.). This, again, is similar in principle to a diabetic diet with restricted protein. A third, and perhaps the best, alternative is to restrict the fat more than the carbohydrate, because the latter best prevents loss of body protein, and consequent loss of strength. A diet

which includes a moderate amount of carbohydrate, especially in the form of green vegetables, has also the advantage of bulk or ballast. Thus, in constructing a dietary it is usual to restrict either the fat or the carbohydrate predominantly. For the reasons given the former alternative is preferable, though in special instances the patient's preferences may be consulted.

INDICATIONS.

The necessity for reduction and the character of the treatment will depend upon the type and degree of obesity, and upon personal and social factors. In the beginning we may assume that a case is of the exogenous type, but if there is no loss of weight after two or three weeks of strict diet (20 instead of 35 or 40 calories per Kg.), an endogenous element should be suspected.[36] The amount of restriction will depend upon the degree of obesity. Von Noorden classifies cases in which the weight exceeds the normal by 5 to 15 kilograms as mild, by 15 to 25 kilograms as moderate, and over 25 kilograms as of high degree, and recommends four-fifths of the normal diet in the first instance, three-fifths in the second, and two-fifths in the third. Thus given a normal requirement of 2500 calories, 2000, 1500 and 1000 calories would be indicated in the several varieties. As a rule, a slow reduction of 1 to 2 pounds a week over a prolonged period is preferable to a more rapid loss of weight. The rapid method usually requires rest in bed at the beginning, and is suitable for patients with an extreme degree of obesity, or for those who desire to obtain quick results. Many persons either do not have the strength of mind or lack suitable facilities for carrying out a prolonged course of treatment, but are willing to submit to periodic terms of deprivation in a hospital or at same watering-place. Facilities for such forms of treatment are less available here than abroad.

In the treatment of a particular case we should determine by the use of the tables and formulæ, with due attention to build and muscular development, what would constitute a normal weight for the patient. A theoretic diet can then be calculated which should make the patient lose a pound or two a week on the average. A careful study of the patient's pre-

vious diet is helpful in outlining such a *régime*. Using this preliminary diet as a basis, further restrictions may be instituted if required. It is wise in a prolonged course of treatment to interfere with the patient's habits as little as circumstances will permit. On the other hand, more rapid results are sometimes attainable by radical modification of the diet. In accordance with the above principle a diet for Americans should consist of three meals, with the possible addition of afternoon tea. Dinner may be either in the middle of the day or at night, according to the patient's previous habit. If breakfast and supper (luncheon) are standardized, the increase (or decrease) of the diet is simplified. It is simpler to construct these diets by the caloric method, which, like the percentage method in infant feeding, allows of a more uniform increase and decrease. Unless dependable formulæ are used (see below) there may be doubt as to the accuracy of values obtained (as these must usually be) by calculation, not by actual analysis. It is quite possible, indeed, to use a purely empiric method, provided that one assures one's self that necessary elements are not cut too low. Whenever possible the food should be weighed at the table. (Sufficiently accurate scales may be obtained for about $15. If the pans are not movable, small papier-maché plates will be found useful, as they may be balanced against each other, and so do away with unnecessary calculations. Still more convenient scales, which may be turned back to zero, after each addition of food, are on the market.) This is of special value during the first few weeks of treatment as an educational matter. After that the patient's judgment will be sufficiently accurate for practical purposes.

The tables which follow were calculated to yield approximately 1000, 1500 and 2000 calories, respectively, or, in other words, amounts suitable for the hypothetic patient with a normal weight of 70 kilograms, and a high, moderate, or slight degree of obesity. In each instance the number of grams of protein, fat and carbohydrate is stated. The data were largely obtained from Locke's convenient tables ("Food Values"), but the simple factors 4, 9 and 4 were used for calculating the caloric values (per Gm.) of protein (P.), fat (F.) and carbohydrate (CH.):

I.

(Additional articles under IV, lists 1 to 4).

BREAKFAST.	Remarks.	Gms.	P.	F.	C-H.	Cal.	
Oranges	½	125	0.7	0.2	10.6	46	See list 1.
Egg	1	50	6.6	6.0	...	80	
Butter		15	0.2	12.8	...	116	
Roll, French	1	39	3.3	1.0	21.7	109	
Coffee		100					
Milk (hot)		100	3.5	4.0	4.5	68	"Café au lait."
Saccharin	2 gr. (if desired).						
			14.3	24.0	36.8	419	
LUNCH OR SUPPER.							
Tea and lemon		200					
Saccharin	2 gr. (if desired).						
Cold veal		50	14.2	0.6	...	62	See list 2.
Salad:							
Lettuce		50	0.6	0.1	2.0	11	See list 3.
Apple	½	75	0.2	0.2	8.0	34	See list 1.
Almonds		5	1.1	2.7	0.8	32	
French dressing		11	...	8.0	...	72	
Calf's foot jelly		50	2.2	...	8.7	44	See list 5.
			18.3	11.6	19.5	255	
DINNER.							
Bouillon		120	2.6	0.1	0.2	13	
Celery		50	0.5	tr.	1.6	8	See list 3.
Roast beef, lean		100	23.3	1.7	...	110	See list 2.
Onions		100	1.2	1.8	4.9	41	See list 3.
Spinach		100	2.1	4.1	2.6	56	See list 3.
½ egg (hard)		25	3.3	3.0	...	40	
Cottage cheese (without cream)		20	4.2	0.2	...	19	
Stewed strawberries (without sugar)		100	1.0	0.6	7.4	39	See list 1.
			38.2	11.5	16.7	326	
Totals			71.8	47.1	73.0	1000	

II.

(Additional articles under IV).

BREAKFAST.	Remarks.	Gms.	P.	F.	C-H.	Cal.	
Apple	1	150	0.5	0.5	16.2	72	See list 1.
Eggs	2	100	13.2	12.0	...	160	
Butter (ball)		15	0.2	12.8	...	116	
Roll, French	1	39	3.3	1.0	21.7	109	
Coffee		100					
Milk		100	3.5	4.0	4.5	68	
Sugar, cube		7	...	...	7.0	28	"Café au lait."
			20.7	30.3	49.4	553	

Lunch or Supper.	Remarks.	Gms.	P.	F.	C-H.	Cal.	
Tea and lemon		200					
Sugar, cube		7	...	...	7.0	28	
Cold chicken		100	32.1	4.4	2.1	176	See list 2.
Asparagus		100	2.1	3.3	2.2	47	See list 3.
French dressing		11	...	8.0	...	72	
Toast, ½ slice		10	1.2	0.2	6.1	31	
			35.4	15.9	17.4	354	
Dinner.							
Oysters	6	85	5.3	1.0	3.2	43	
Celery		50	0.5	tr.	1.6	8	See list 3.
Mutton, boiled, lean		100	30.9	4.5	...	164	See list 2.
Squash		100	1.4	0.8	13.6	67	See list 3.
Spinach		100	2.1	4.1	2.6	56	See list 3.
½ egg		25	3.3	3.0	...	40	
Swiss cheese		20	5.5	7.0	0.2	86	
Cantaloupe	¼	232	0.7	...	10.5	45	See list 1.
			49.7	20.4	31.7	509	
Totals			105.8	66.6	98.6	1416	

III.

By the addition of a roll and butter (225 calories), a potato (126 calories), and a simple dessert such as soft custard (131 calories) to the dinner, the diet (II.) may be brought up to approximately 2000 (1898) calories. Indeed, it will be easy to exceed that limit if care in the choice and weighing of the food be relaxed.

The following lists* give the weight and caloric values of various foodstuffs which may be substituted for the articles given in the diets; also articles that should be avoided or only used by special permission:

IV.

1. *Fruits.* (Fruits may be stewed without sugar or be taken raw). Apple, 150 grams = 72 calories; blackberries, 100 = 59; ¼ cantaloupe, 230 = 45; cherries, 100 = 76; currants, 100 = 59; ¼ grapefruit, 150 = 69; grapes, 75 = 56; huckleberries, 100 = 76; ½ orange, 125 = 48; peach, 125 = 42; ½ pear, 75 = 42; pineapple, 50 = 44; plum, 35 = 29; raspberries, 50 = 28; strawberries, 100 = 40; watermelon, 300 = 39.

* Also see list at end of chapter on Metabolism.

To be avoided: Bananas, dried fruit, preserves, jams, jellies and marmalade.

2. *Meats, Fowl and Fish.* Beef, roast, very lean, 100 grams = 111 calories; beef "round," fat removed, 100 = 185; sweetbread, 80 = 135; chicken, roast, 100 = 180; mutton, boiled, lean, 75 = 126; mutton-chop, lean, 100 = 135; pork (lean ham), 33 = 93; cod (boiled), 100 = 98; haddock (boiled), 100 = 108; halibut (boiled), 100 = 121.

To be avoided: Beef tenderloin, tongue, capon, lamb, bacon and pork in general; turkey, goose, duck, bluefish, mackerel, salmon, and fat meats in general. Also hashes, croquettes, sausages, fried meat, etc.

3. *Vegetables.* Artichokes, 100 grams = 27 calories; asparagus (canned), 125 = 23; string-beans, 60 = 13; beets, 70 = 29; beet-greens, 100 = 54; cabbage, 100 = 5; carrots, 100 = 18; cauliflower, 100 = 7; celery (raw), 50 = 9; corn (canned), 100 = 101; corn (green), 100 = 100; cucumbers, 50 = 9; dandelion-greens, 100 = 63; mushrooms (raw), 50 = 23; onions, 100 = 42; parsnips, 100 = 10; baked potato (special), 100 = 114; squash, 100 = 69; spinach, 100 = 57; tomatoes (canned), 70 = 16; tomatoes (raw), 100 = 23; turnips, 100 = 4.

To be avoided: Beans in general, corn, mushrooms (except raw), peas, potatoes (sweet or white) in all forms. Also hominy, rice, and macaroni (often used as "vegetables").

4. *Soups and Raw Shellfish.* Beef-soup, 120 grams = 32 calories; bouillon, 120 = 13; consommé, 120 = 14; Julienne, 120 = 16; vegetable (canned), 120 = 17. Also raw clams (round) (6), 100 = 47; raw oysters (6), 85 = 44.

To be avoided: Bean, chicken, green and mock turtle, oxtail, pea and tomato-soups. Also cream-soups, stews and chowders.

5. In addition to articles specifically prohibited above, avoid: (1) All fatty food—butter, cream, olive oil, etc., and all articles prepared with fat—fried food, pastry, rich sauces, puddings, etc. (2) All starchy food, including breads, cereals, desserts, thickened sauces and gravies. (3) Sugars and all sweets—candies, cakes and desserts. Condiments and sauces are objectionable in proportion to their content in sugar or oil.

In mild cases gelatin (calf's foot jelly), custards, etc., prepared with a minimum of sugar may be allowed.

The patient should be weighed once a week, and always at the same hour. This should be several hours after breakfast, with bladder and bowels empty. On the basis of a gain or loss of weight the diet should be readjusted. No mention has been made of fluid restriction, because this is not indicated in simple obesity. On the day before weighing, and in any event, on the day of weighing, a uniform amount of fluid should be ingested. Following the active treatment, which may last from a few weeks to several months, a diet not more than sufficient to keep the weight stationary should be instituted. If, in spite of this, there is a tendency to gain, fast days are indicated (Boas) once a week or less frequently. On these days Boas allows 100 grams of black bread, 2 hard-boiled eggs and an apple. Others limit the food to a liter of milk.

SPECIAL DIETETIC MEASURES.

In cases complicated by cardiovascular or renal disease, as well as in gout and diabetes, special modifications are desirable. In gout the protein should not exceed 1 gram per kilogram, and foods rich in purin should be avoided. A moderate restriction of protein is also advisable in nephritis. In cases with edema, fluid restriction is of great importance, and this, as well as salt restriction (particularly useful in nephritis), is provided for in some of the special dietaries.

Karell's diet, originally designed for the removal of dropsy,[37] has been modified by Moritz, Rosenraad[38] Jacob, and others for cases of obesity. This modified diet consists essentially of 2000 mils of milk for a person with a metabolism of 2800 to 3000 calories, and represents approximately 20 calories per kilogram. It contains very little salt. A potato, representing 100 calories, is frequently added to protect the protein.

After a variable time of a few days to a week or more, transition is made to a diet poor in fat, consisting of lean meat, green vegetables, stewed fruit, and 4 to 5 ounces (125 to 150 Gms.) of graham (black) bread. The fluid is restricted to 1 quart (1000 mils). Other diets recently suggested for

obesity are the vegetarian,[39] desirable on account of the variety offered, and the potato diet (Rosenfelt), which is effective for precisely the opposite reason, for its monotony destroys any desire for excessive food. Both of these diets are bulky, and in this sense satisfying. The general principles underlying the classic diets of Oertel and Ebstein have already been mentioned.

A modified form of vegetarian diet, recently prescribed at a prominent watering-place, consisted of 4 glasses of milk (1 qt.), 2 glasses of buttermilk (1 pt.), 4 glasses of Vichy (1 qt.), and 3 baked potatoes (1200 to 1350 calories). In this case a large amount of fluid was allowed, probably because of a "gouty" element in the case.

DRUGS.

The drug treatment of obesity may be dismissed in a very few words. The exogenous form requires no drug treatment other than symptomatic measures indicated by special complications, such as anemia, constipation, intestinal flatulence, and the like. These should be treated according to the principles laid down elsewhere in this work. In the endogenous form of obesity, *dried thyroids* may be used with excellent effect. Inasmuch as large doses have been known to bring on symptoms of hyperthyroidism (rapid pulse, nervousness, loss of weight and strength), which are very difficult to control, the dosage at the beginning should be very small. One-half grain (0.03234 Gm.) three times a day will be ample, and may be cautiously increased up to the pharmacopœial dose, 1½ grains (0.1 Gm.). This drug treatment should be used in connection with the dietetic treatment, as recommended for simple obesity. Other drugs, official or proprietary,[40] have been suggested for this condition, but none of them can be recommended.

HYDROTHERAPY.

The daily intake of water is often restricted in obesity, first for the purpose of diminishing edema,—a use which is well founded,—and, secondly, for the purpose of reducing weight, independent of edema. Although the weight may be reduced temporarily by this measure, it would seem in the

latter instance to subject the patient to the danger of insufficient elimination with no compensating advantage. Prof. Kisch, of Marienbad, advises mineral waters containing Glauber's salts. He is uncertain whether they act by increasing CO_2 consumption and fat utilization, or merely by interfering with absorption, on account of the free movement of the bowels. There seems to be no valid indication for these waters other than for constipation and flatulence. Frequently abdominal distention is due in part to gas, and may be relieved by suitable treatment.

Steam, hot-air, and electric-light baths, usually followed by a cool douche, shower or plunge, and massage are widely used to reduce flesh. Undoubtedly a temporary reduction of weight may be obtained by this method, but this is due solely to the loss of water, and is of no value in the absence of renal complication. In cases with nephritic edema, a similar but more permanent reduction may be obtained by salt restriction. Baths have, however, undoubted collateral benefits, improving the condition of the skin, increasing peripheral circulation, increasing elimination, and stimulating the heart and the nervous system. Prolonged cold baths, combined with rubbing and exercise, are theoretically rational, since it is evident that a certain amount of heat, representing food consumption, may thus be abstracted. Gärtner[41] advises rowing movements performed while in the cold bath, using elastic-rubber tubes (these may be attached to the faucet) to furnish the resistance. The Nauheim baths are used in obesity complicated with heart disease, primarily to improve the tone of the heart-muscle. (See Diseases of the Cardiovascular System, Vol. II.) Baths combined with vigorous resistance movements, graduated according to the strength of the patient's heart and general musculature are now being used extensively in our large cities for weight reduction in gouty, overfed, and underexercised business men. These may be of great benefit, but should not be undertaken without preliminary examination of the heart by a physician.

Passive exercises are most suitable for the aged, and those with weak heart. Deep breathing and active movements without apparatus (Swedish movements, setting-up drill) are useful if systematically carried out. Like all indoor gymnastics,

they soon become irksome. More elaborate gymnastic equipment, like the Zänder apparatus, is not usually available except in a few large cities. Outdoor exercise has the great advantage of offering variety, interest, and the opportunity of deep breathing of fresh air. For middle-aged adults the most useful and available exercises are walking, climbing and golf. The automobile is undoubtedly conducive to obesity, but may be used with signal advantage, since it makes walking and climbing possible even for those who live in closely built-up towns. Golf has hitherto been a sport for the well-to-do, but with the rapid introduction of public links this pastime is now becoming available for the general public. Swimming is another valuable exercise. Finally, tennis and baseball should be mentioned for the young and vigorous.

General massage has little effect in reducing obesity, but many competent clinicians believe that local massage of the abdomen or hips may favor a more desirable distribution of the adipose tissue. Bergonie[42] has devised an electrical apparatus by which muscles in various regions may be passively exercised without discomfort to the patient. In this way it is claimed that the muscles of the hips, for example, may be rapidly reduced. Mechanical massage, by means of vibrators and rollers, has been employed for the same purpose.

The "Spa" treatment is much in vogue abroad, Homburg, Kissingen, Marienbad and Vichy being a few of the better known resorts. In this country patients at "baths" are under a less close surveillance. There are suitable facilities for treatment at Saratoga Springs, N. Y.; Hot Springs of Virginia; White Sulphur Springs, West Virginia, and elsewhere. Under strict treatment in such resorts patients may lose half a pound a day or more. Such a rapid loss is seldom permanent.

GOUT.

Gout,[43] a metabolic disorder of uncertain etiology, is almost uniformly associated with an increased amount of uric acid in the blood, and in long-standing cases with uratic nodules in the subcutaneous tissues, or in and about the joints. This underlying anomaly is more or less permanent, but may be punctuated: by acute "fits" of arthritis (arthritis

uratica), usually attacking the joints of the lower extremity, and particularly those of the great toe (podagra); by acute or subacute attacks of polyarthritis; or by a variety of irregular manifestations. While the view that gout is primarily a disturbance of purin metabolism is generally accepted, some authorities consider it a toxemia of hepatic and intestinal origin, and regard the uric acid deposits as merely incidental. The relation of the uric acid to the overt manifestations of the disease is obscure. Most theories connect it in some way with the precipitation of monosodium urate in the joints and elsewhere. The popular idea of uric acid poisoning is based on the erroneous assumption that uric acid is toxic. Many conditions are attributed to the uric acid diathesis, which are identical with those at times ascribed, with equal lack of discrimination, to neurasthenia or intestinal autointoxication.

The *uric acid diathesis* is a term that has very properly fallen into disrepute and has long formed a cloak for ignorance. The condition which the term was intended to designate is one in which the patient suffers from one or more of the various and many symptoms met with in arthritis in general, including neuritis and headache.

Gout is one of the few diseases in which the uric acid metabolism is believed to play a causative rôle, and in general the term uric acid diathesis is a misnomer.

Typical podagra is relatively rare except in England, because in no other country have widespread conditions of leisure and luxury existed for so many generations. On the other hand, the statistics of Futcher[44] indicate that among the general population, exclusive of the upper classes, gout is almost as common in the United States as in England. In the latter country there is undoubtedly a disposition to attribute too many complaints to a gouty origin, and in this country we have fallen into the same error, having labeled some cases of arthritis and rheumatism—particularly in those past middle age—as subacute or chronic gout. The tendency at present is to limit the term rheumatism to the acute specific infection (rheumatic fever). Some of the cases of acute podagra occasionally seen are associated with renal sclerosis and a history of alcoholism. In these patients there is usually no hereditary element.

Of heredity, an important factor in England, Roberts[45] says that "fully three-fourths of the cases of gout, occurring among the easy classes, can be traced back distinctly to a gouty ancestry." In many instances, however, English authors do not seem to be very critical in their judgment of what constitutes a history of the gout. Thus asthma, sore throat and eczema are classified as gouty, often on seemingly insufficient grounds. Patients, moreover, are very prone to attribute their own maladies and those of their forbears to this aristocratic disease. It may be safely asserted that the hereditary factor is relatively unimportant in this country.

Gout is a disease of middle life—after 40—and is much more common in men than in women. The incidence of gout in middle-aged men is accounted for by their special liability to overindulgence in rich food and malt liquors. In women the menstrual discharge is thought by many to have a prophylactic value. All authorities agree that the deleterious effect of alcoholic beverages is not in proportion to their alcoholic content. Distilled liquors are more likely to lead to cirrhosis of the liver, while wines, particularly those which have been fortified or refermented (port, sherry, burgundy, champagne), ales and porter are conducive to gout. A nitrogenous diet, *per se,* provided that it does not contain an excess of purins, is not now considered injurious in cases of pure gout (*i.e.,* without renal involvement).

In England lead-workers are unusually predisposed to gout, but this coincidence is not common in this country. Brewery workers, stewards, butlers, and others who have free access to rich food and alcoholic beverages often develop this disease. Exercise may serve to neutralize bad dietetic habits, but is by no means a panacea. In persons predisposed to gout some exceptional influence, such as trauma or cold, may induce an acute or subacute manifestation; thus, a "gouty" arthritis or neuritis may seemingly follow overstrain or slight injury. Emotional causes are also credited with a similar effect. Sir William Roberts believed that a course of treatment with waters containing sodium salts was very likely to induce an attack of acute podagra. Finally we must emphasize the association of gout and chronic nephritis. The latter causes a retention of uric acid in the blood.

As stated in the definition, gout is associated with the deposition of salts of uric acid in the joints and elsewhere. The metatarsophalangeal joints of the great toe are those most often attacked. The instep, ankles, knees, hands, and wrists are affected approximately in the order indicated. The elbows, shoulders and hips are rarely attacked, and the articulations of the jaw and the sternoclavicular joints almost never. Abarticular deposits are described in the following situations: the rim of the ear, the tendons, the aponeuroses, the skin of the palms and soles, the eyelids, nose, vocal cords, cranial and spinal dura and pia mater, the sclerotic coat of the eye, the fibrous sheaths of nerve-trunks, the aortic valves.[46] The nodules on the helix of the ear, which are by far the most common, at first contain a whitish fluid, but subsequently are converted into minute, solid, whitish nodules, which may be readily removed for examination. Osler warns us not to mistake the Darwinian tubercle, which corresponds to the primitive tip of the ear, for a gouty tophus. In chronic gout the tophi may be the only overt manifestation of the disease. In certain cases of so-called deforming gout, there are irregular "chalky" deposits or nodes in or about the joints of the hands and elbows, and in aggravated examples of such deformities the hands resemble fantastic roots or tubers.

With the *x*-ray the gouty nodules cast a very faint shadow. The heads of the phalanges and other affected bones show characteristic circular areas of absorption from a millimeter to a centimeter in diameter, which are surrounded by dense ring-like borders. Pathologically, the tophi as well as the areas of rarefaction are found to contain crystals of monosodium urate. In joints affected by gout the uratic deposit is found close to the surface of the cartilage, but separated by a thin endothelial layer from the joint-cavity. In the tendons and other connective-tissue structures the uric acid salts are deposited in the interstices, and sometimes may be dissolved out without leaving any gross alterations. Necrosis and other pathologic changes—whether primary or secondary is a matter of dispute—are frequently seen in connection with gouty lesions.

Uric acid is the product of the destructive metabolism (catabolism) of the nucleins, not of the proteins in general.

By a process of deamidization and oxidation brought about by specific ferments, adenin and guanin are split off from nucleic acid, and from these, in turn, hypoxanthin and xanthin, from which uric acid is derived. These five substances are spoken of as purin bodies, because of their supposed relation to a hypothetic substance known as purin. The formulæ follow: purin ($C_5H_4N_4$); adenin ($C_5H_5N_5$), 6-aminopurin; guanin ($C_5H_5N_5O$), 2-amino- 6-oxypurin; hypoxanthin ($C_5H_4N_4O$), 6-oxypurin; xanthin ($C_5H_4N_4O_2$), 2-6-dioxypurin; uric acid ($C_5H_4N_4O_3$), 2-6-8-trioxypurin. The exogenous purins are those derived from the nuclein of ingested foods; the endogenous, from the nucleins of the patient's own body cells. The excretion of the former can be readily controlled by dietetic treatment, while the excretion of the latter is peculiar to the individual, and cannot be controlled in the same way. Foods distinctly harmful on account of their high content of purins are those rich in nucleins: glandular organs (liver, sweetbreads, kidneys), broths and meat-soups. Meats in general, including fowl and fish, and leguminous vegetables are harmful in a minor degree. Milk, butter, eggs, cheese, most cereals, fruit, nuts, and green vegetables are nearly, or quite, purin-free.

As already stated (see p. 451), uric acid is a normal constituent of the blood (on a purin-free diet from 0.5 to 2.9 mgs. per 100 Gms. of blood). Pratt[47] and others have shown that uric acid is present in moderate excess (average 3.7 mgs. per 100 Gms. of blood) in chronic gout, and in more considerable amounts (*e.g.*, 5.7 mgs.) at the beginning of an acute attack. Excess of uric acid in the blood is spoken of as uricemia. Walker Hall[48] differentiates: (1) temporary or dietetic uricemia (after food rich in purins); (2) functional uricemia (in leukemia and pneumonia); (3) retention uricemia (in renal disease and uremia); (4) permanent uricemia (in gout). He states that in gout associated with contracted kidney as much as 20 milligrams of uric acid per 100 mils of blood have been found.

The urine of chronic gout shows no constant variation in uric acid excretion, though most investigators have found a diminished output. In an acute attack uric acid is diminished in the beginning and increased as the attack subsides. With

this there is a parallel change in the excretion of phosphoric acid. In chronic gout it is common to find the urine with a low specific gravity, a faint trace of albumin, and a few casts. In an acute attack there are the high color, high acidity, and precipitation of urates common to fevers. Neither the blood nor the urine of gout shows any constant diminution of alkalinity or increase of acidity, in spite of the popular, and even professional opinion to the contrary.

A typical acute attack of the gout comes on with little or no warning, very often in the middle of the night. Occasionally prior to an attack the patient may have suffered from digestive disturbances or vague joint-pains. The affected joint, which in more than half the cases is that of the great toe, becomes red, swollen, hot and exquisitely tender. These symptoms may be so intense that abscess may be suspected; indeed, in one case that came under observation a physician urged "immediate incision" of the supposed "felon" (in this instance a finger was affected). The swelling persists from a few days to a week or more, and in the early stages is accompanied by moderate fever and associated symptoms: rigors, sweating, loss of appetite, concentrated scanty urine, etc. The symptoms frequently remit during the day, only to recur with increased severity at night. In addition to the original focus, other joints may be affected, particularly those of the tarsus and ankle on the same or on the opposite side; less often the joints of the upper extremities. At the height of the swelling the tissues surrounding the affected joint may pit on pressure. This and the subsequent desquamation are regarded as suggestive, from a diagnostic point of view. A person who has had an attack of gout frequently suffers subsequently from annual or semiannual visitations of the malady. This tendency to recurrence, although not a constant factor, is characteristic. Recurrences may, or may not, be prevented by careful dietetic treatment. An attack such as that described is most often seen in the hereditary type, and in these instances there may be little or no evidence of nephritis. On the other hand, in chronic diffuse nephritis it is not unusual to see quite similar manifestations, not rarely following an attack of uremia. In these patients retention uricemia is obviously a more important factor than in the

uncomplicated disease. A third variety of acute or subacute gout is the polyarticular form. This may be difficult to differentiate from acute articular rheumatism.* [49] The age of the patient, the history, the laboratory findings, and, most important of all, the presence of tophi will point to the diagnosis.

Subjects of acute gout in time present the evidence of the chronic affection: tophi in the ears, about the smaller joints and elsewhere. They also may be victims of recurrent sore throat, asthma, and other conditions to which gout predisposes. The ultimate prognosis of gout depends largely upon the complications, and in particular upon the condition of the kidneys and of the vascular system.

Chronic gout may be associated with the acute form or pursue its course without typical acute attacks. The victims of the gouty diathesis are traditionally inclined to obesity; they are florid and thin-skinned, with a tendency to vascular hypertension. The high blood-pressure is probably due to renal fibrosis, as we see patients with distinct gouty tophi and normal urine whose blood-pressure is normal or subnormal. Gouty patients suffer from a great variety of affections, attributed with more or less probability to the underlying vice of metabolism. When such symptoms occupy the foreground we speak of the condition as irregular gout. The following is a brief systematic enumeration of some of these affections mentioned by authors, but in many cases the etiologic link is not very evident:

Neuritis and migraine are common in gouty subjects, and are relieved by measures similar to those useful in gout. Undoubtedly other toxemias may produce similar manifestations. The cases of "neuritis" attributed to this cause have not shown pronounced atrophy or complete loss of reflexes. It is often difficult to exclude fibrositis. Vertigo, irritability, mental depression, and insomnia are common functional symptoms. Apoplexy is the result of the accompanying vascular sclerosis.

Tachycardia, precordial oppression, and anginoid pains are common in this disease, and may be related to a hypo-

* Brugsch[50] describes a polyarticular form which closely simulates rheumatoid arthritis.

thetic toxemia. High blood-pressure, sclerosis of the arteries, myocarditis, hypertrophy and dilatation of the heart are the sequels or concomitants of the complicating nephritis, rather than of the uric acid diathesis itself. Phlebitis is described as a common symptom.

The connection of gout with affections such as laryngitis, bronchitis, asthma, pharyngitis and hyperchlorhydria is more problematic, and certainly of very little practical importance in American practice. The association of chronic nephritis with gouty deposits in the joints and with frank gout is indubitable. It is more likely that the renal insufficiency precipitates the gouty attack by interference with elimination (retention uricemia), than that a gouty toxemia initiates the renal mischief. Fibrositis or myalgia is a distinct affection to which cold and trauma seem to predispose. It may frequently simulate neuritis. It is quite possible that in some cases the gouty diathesis may be a predisposing factor.

Acute articular rheumatism, various forms of infectious arthritis, and arthritis deformans of several types are to be carefully distinguished from gout. We should suspect gout as a possible cause in any case of acute "rheumatism" which attacks the lower extremities predominantly, and is unaccompanied by endocarditis. The suspicion is strengthened if the patient be no longer youthful, and salicylates fail to have their customary happy effect. Arthritis deformans, on the other hand, is more likely to be considered gout than the reverse. Persistence of the symptoms, the spindle form of the joints, and the development of atrophy, or of exostosis, are a few of the signs which should suggest the correct diagnosis. Heberden's nodes are never to be considered as a manifestation of gout, although a gouty person may present them.

Eczema is so common a complication of gout that the latter must be admitted as one of its causes. In this disease it occurs most frequently about the face, as a dry, scaly eruption. Psoriasis and other skin diseases have also been attributed to a gouty origin. Schamberg found that patients with psoriasis retained nitrogen, and were benefited by a diet poor in nitrogen.

Rarely as a result of treatment, or spontaneously, an acute articular gout may suddenly disappear, and be succeeded by

severe cerebral, cardiac, or gastrointestinal symptoms—the so-called retrocedent gout. Cold applications to the joints (probably without good reason) are sometimes credited with this dire effect.

TREATMENT.

Certain general principles apply to the treatment of the gouty diathesis, independently of its overt manifestations, acute, chronic or irregular. Whatever our theories as to the ultimate cause of gout, its uniform association with defective uric acid metabolism is the one outstanding fact upon which all may agree. This fundamental, if not primary, defect should, therefore, be the first consideration in treatment. If we follow the analogy of diabetes our aim should be to strengthen the impaired functions by *functional* rest. There are also other disturbances of metabolism, and of the various systems, renal, cardiovascular and digestive, which will uniformly require investigation, and probably therapeutic attention. Gouty subjects suffer from faulty digestion, unstable metabolism and imperfect elimination. Their margin of safety is, therefore, limited in these and other directions, and they must be subjected to corresponding limitations. In all cases individualization is most important.

The condition of many of the organs and functions may be arrived at by special functional tests, as well as by the ordinary clinical laboratory methods. A careful examination of the urine, with special references to low specific gravity, traces of albumin, and casts may be supplemented by estimation of the blood-pressure, phenolsulphonephthalein elimination in the urine, and total non-protein nitrogen of the blood (method of Folin). If facilities for nitrogen estimates are not available, the urea of the blood may be determined by the urease method. In this way a good idea of the functional capacity of the kidney may be obtained. Folin has devised a method for the quantitative elimination of uric acid in the blood, but this is rather too complicated for clinical purposes. Simpler and more useful, perhaps, is the estimation of the uric acid elimination in the twenty-four-hour specimen of urine, first on a purin-free diet, and then on a diet rich in purins. The normal excretion on a purin-free diet is 0.4 to

0.6 grams (equivalent to 0.11 to 0.17 Gms. uric acid N), while added purins are rapidly eliminated. In the gouty the daily excretion may be diminished, and added purins are very slowly eliminated, generally requiring more than two days. It is also well to test the patient's tolerance for sugar as described under "Diabetes"—by the administration of 100 grams of glucose. The gastric and intestinal functions should be investigated by the usual test-meals, and by the Schmidt diet. This will reveal alterations in acidity, faulty digestion of meats, fats and starches, etc.

DIET.

The diet should be arranged to meet the following indications: (1) Faulty elimination of uric acid and purins, to be met by a diet free, or nearly free, from purins. (2) Impaired nitrogenous excretion, depending on the state of the kidney; to be met by moderate or strict limitation of protein generally. (3) Glycosuria, usually successfully met by restriction of sugars alone. (4) Tendency to overnutrition; to be met by a modified obesity diet. (5) Digestive disturbances, hyperacidity, flatulence and constipation; to be met by restriction of starches, administration of green vegetables and fruits, and by the exclusion of indigestible articles.

The following table shows the purin content of the principal foodstuffs:

PURIN BASE-CONTENT OF VARIOUS ARTICLES OF DIET ACCORDING TO HESSE.

(The purin bases in this table are calculated as "uric acid." Grams of uric acid in 100 grams.)

Thymus	1.308	Sole	0.137
Liver	0.372	Caviar	0.110
Kidneys	0.320	Oysters	0.217
Brain	0.233	String-beans	trace
Beef	0.175—0.189	Carrots	0.007
Mutton	0.189—0.191	Potatoes	0.019
Veal	0.178—0.189	Asparagus	0.057
Pork	0.181—0.185	Cabbage	0.078
Chicken	0.186	Green peas	0.079
Venison	0.182	White beans	0.098
Squab	0.154	Pea meal	0.108
Trout	0.213	Wheat meal	0.116
Salmon	0.201	Rye meal	0.096
Pike	2.222	Milk	0.010
Cod	0.131	Eggs	trace

The most important purin-free, or nearly purin-free, articles of diet are: milk, butter, cheese, white bread, tapioca,

rice and other cereal foods in which the outer coating of the grain is removed, fruits, nuts, and green vegetables (except asparagus, cabbage and green peas). Except in very rigid diets root vegetables are also allowable, particularly carrots and turnips. Dried beans, peas and lentils are comparatively rich in purins. Experiments of Luff (*l.c.*) seem to show that spinach, Brussels sprouts, cabbage, French beans, celery and turnips are of positive benefit, since their mineral constituents tend to increase the solubility of sodium biurate in vitro.

In an acute or a subacute attack, or during convalescence, a diet as nearly as possible purin-free should be prescribed. In the febrile period this may consist largely of milk, but subsequently any articles from the foregoing list may be selected. In convalescence, certain meats may be allowed once a day.

The following articles should be avoided by the gouty:[51]

Rich meat soups—ox-tail, turtle, mock-turtle, kidney, mulligatawny, hare, giblet.

Salmon, mackerel, eels, lobsters, crabs, mussels, salted fish, smoked fish, preserved fish, tinned fish. Duck, goose, pigeon, high game. Meats cooked a second time.

Hare, venison, pork, lean ham, liver, kidney; salted or corned meats, pickled meats, preserved and potted meats; sausages; all highly seasoned dishes and rich sauces.

Tomatoes, beet-root, cucumbers, rhubarb, mushrooms, truffles.

Rich pastry, rich sweets, new bread, cakes, nuts, dried fruits, ices, ice-cream.

In chronic or irregular gout, or in the intervals between acute attacks, meat or fish may be allowed once a day, but organs unusually rich in purins, such as liver, kidneys, sweetbreads, and, perhaps, leguminous vegetables, should be forbidden. Coffee, tea and chocolate should be used in great moderation, or not at all. Coffee prepared with hot milk in the French fashion (*café au lait*), or freshly steeped weak tea, may be permitted to those who are dependent on their morning beverage. Caffein-free coffee, and various coffee substitutes are also admissible. Caffein and theobromin are methyl purins, but the best evidence at hand indicates that they are eliminated unchanged, and do not give rise to uric acid as do the purin bases. It is well, however, to exclude

coffee and tea "on suspicion," particularly as their omission may be justified on other grounds.

The protein need not be unduly restricted in gout; the amount allowed is to be regulated in proportion to the functional capacity of the kidneys. In the ordinary case protein may be safely reduced to 75 grams a day, or, with positive evidences of renal insufficiency, to 50 grams or less. Sodium chlorid may be advantageously limited if there be any tendency to edema. As already stated, Sir William Roberts believed that sodium salts, in general, were deleterious in gout, and might cause a precipitation of sodium urate. Broths, meat-soups and gravies, which are rich in nitrogenous waste products, should be avoided, particularly so if the case be complicated by nephritis.

Fats are well borne, and need only be restricted if there is a tendency to obesity, or if they interfere with digestion. The latter contingency is only likely when they are employed in conjunction with carbohydrates, as in pastry, fried foods, and as fat meats and fish. The fat prevents access of the digestive juices, and interferes indirectly with digestion. Occasionally there may be a subicteric hue and clay-colored stools, which may show free fat under the microscope. In such cases fats may be temporarily restricted.

Carbohydrates are often a cause of difficulty in gouty persons, quite aside from the production of glycosuria. Dyspepsia is a common, virtually an invariable, accompaniment of gout, and is more often of an "amylaceous" type. With the clinical symptoms (heartburn, water-brash, flatulence), and the laboratory evidences (increased acidity, poor starch digestion) of hyperchlorhydria, amylaceous articles should be cut down. (In aggravated cases of "flatulent" dyspepsia the "Saulsbury" diet, consisting solely of lean beef and hot water, is occasionally used.) The most appropriate forms in which they may be allowed are dry toast, rusks, zwieback, a moderate amount of thoroughly cooked cereal (preferably without husks), baked potatoes, and finely divided green vegetables. Acids and acid fruits should be tabooed; also articles which tend to flatulence, such as baked beans and boiled cabbage. Condiments, salt (in excess), pepper, mustard and other spices, meat-broths and soups, rich sauces and relishes

should be restricted, because they tend to stimulate acidity. Olive oil, cream, butter, tender meat, fish, or fowl are usually easily digested in the stomach, and may be allowed, in spite of the gouty constitution. Boiled meats are preferable to roasted, because a certain amount of extractives are removed in this way. In constipation fruits and fruit juices, green vegetables, olive oil, and the coarser breads and cereals may be employed, if not contraindicated by any of the above conditions. The treatment of glycosuria is considered under "Diabetes" (*q.v.*).

In gout free administration of water, either as ordinary soft potable water, distilled water, or mineral water (Poland or Evian), is advisable, except in cases of cardiac insufficiency, or in renal cases associated with dropsy. The water is preferably taken between meals, as well as at night and before breakfast. In some cases it may be preferred hot. "Soft drinks" are not to be recommended, as they are often sweet, and upset the digestion. In general, there is no objection to lemonade, weak tea, cider, and nutritious beverages such as milk and plain buttermilk. Alcohol should be forbidden. The harmfulness of alcoholic beverages, as already pointed out, does not appear to be connected with their absolute alcoholic content, or with their acidity. Malt liquors, port and sweet wines are to be unreservedly forbidden. In exceptional cases, as in the aged and infirm, or in those long addicted to the use of alcohol, claret or well-diluted whisky or gin is permissible.

HYGIENIC TREATMENT.

On account of their defective assimilation and elimination, the subjects of the gouty diathesis are more dependent on exercise, baths, climate, and similar aids to health than normal individuals. Moderation in food and drink independently of any special restrictions, regular meals, careful chewing, a plentiful supply of pure water, and attention to regularity of the bowels are essential.

Outdoor exercise is almost always advisable, but its character will depend upon inherent factors such as the patient's previous habits of life, the condition of the heart, and degree of obesity. The same remarks apply here as in obesity; for

the middle-aged, golf is the most suitable exercise, but its beneficial effects are too often neutralized by indulgence in rich food and alcohol. Walking is the most universally available recreation, particularly valuable in cardiac cases. Motoring is, for obvious reasons, probably the worst possible recreation for an able-bodied gouty individual. Sedentary occupations and those involving exposure to lead or inviting to the use of malted beverages are deleterious. In the case of lead-workers a change of occupation should be arranged; for those following other occupations provision of hours for exercise and revision of the habits of eating and drinking will be sufficient.

Climatic treatment, except in association with the use of mineral waters, does not occupy a large place in the treatment of gout. A cool, bracing climate, favorable to outdoor exercise, is preferable for a majority of persons. In foreign countries the treatment at various watering places has been highly developed, and through a combination of factors—regular hours, graduated exercise, carefully supervised diet, free consumption of water, pleasant surroundings and favorable climate—excellent results are obtained. This is especially true of persons who, through choice or compulsion, have led a life of inactivity and high living through the greater part of many years. The mineral constituents of the various springs differ widely, and we must attribute their direct influence on uric acid metabolism to their quantity, rather than their quality. The waters most celebrated for this purpose are those which contain the least mineral constituents. Many of the waters are hot, and this may add to their local and general effects. At all the spas elaborate provision is made for baths, douches, massage, etc. The special value of lithium waters has long since been discounted. Other mineral springs contain substances which are useful in the treatment of various concomitant manifestations and complications of gout. As stated, their direct effect may be harmful, because in most of them there is a predominance of sodium salts. The waters of Vichy are rich in sodium bicarbonate, and are especially indicated in cases with hyperacidity. The Vichy douche massage is a celebrated method of local treatment. The waters of Carlsbad and Marienbad owe their virtue to a com-

bination of sodium chlorid, sodium bicarbonate and sodium sulphate. They are supposed to be especially useful in the congestion of the liver, cholelithiasis and constipation. Another class of mineral springs, much resorted to by the obese and the gouty, and especially indicated in catarrhal conditions of the stomach and intestines, is characterized by their high sodium chlorid content. The best known of these springs are Homburg, Wiesbaden, Kissingen and Baden-Baden. Various sulphur springs, such as Aix-les-Bains, are recommended for their local effects upon the joints, and particularly upon cutaneous manifestations. In this country waters resembling those mentioned are to be found at Poland Springs (very slightly mineralized), Saratoga Springs, Bedford Springs, Hot Springs of Virginia, White Sulphur Springs and elsewhere. At most of these resorts excellent accommodations are afforded, and at many of them competent physicians are available. Elaborate provisions are also made for baths, massage, local treatment and exercise. The discipline, however, is not so good as at the German spas. Most of the visitors are bent on pleasure, and it is difficult for patients to observe a strict *régime*. Many enthusiasts for this form of treatment bolster their faith with certain chemical discoveries. Thus they claim a special efficacy for minute amounts of mineral salts, on account of their high ionization. Special therapeutic efficacy is also ascribed to the radium emanations given off by some mineral waters when freshly drawn. The accessory modes of treatment provided at the various resorts are now equally available in all our large cities, and a physician may have his hydrotherapeutic prescriptions carried out, just as in the case of drugs. Most physicians, however, are so unfamiliar with the general procedures that they are inclined to leave the execution of details to lay hands. With greater experience this fault will doubtless be corrected.

The electric-light cabinet bath is the most generally useful apparatus for promoting elimination. The operators claim that there is some virtue in the light itself, but this seems improbable. Its chief superiority consists in the readiness with which the heat can be controlled. The portable hot-air bath will serve equally well in private practice. When a gentle perspiration has been established, the patient is given

a lukewarm or cold bath. In the young and vigorous, without marked elevation of blood-pressure or cardiac weakness, a cold shower or plunge is employed. Additional stimulation may be given by the Scotch douche, which consists of alternating jets of hot and cold water under high pressure. For the less vigorous a warm rain or tub bath, followed by gentle massage, is safer. Sometimes alternating hot and cold douches, combined with massage, are used on individual joints.

MEDICINAL TREATMENT.

A large number of drugs have been vaunted for their alleged specific effects on uric acid metabolism, some because they were supposed to promote solution of the deposits, others for their effect on elimination. Still others have been prescribed to alter the reaction of the urine, to control pain, and to combat various individual symptoms.

Colchicum [tincture of colchicum-seed, 30 minims (2 mils), and colchicum, $\frac{1}{120}$ grain (0.5 mg.)], has long enjoyed a high reputation for its effect on the pain and inflammation in acute gout. It has no effect on uric acid excretion, and some authors go so far as to attribute its favorable action solely to its purgative qualities. Colchicum is of little value in chronic gout.

Salicylates are less effectual than colchicum in controlling the pain of gout, but they increase the excretion of uric acid to a marked degree. They are useful in subacute and chronic gout, and in irregular gout.

Atophan[52] (phenylquinolin-carbonic acid) and related compounds (isatophan, novatophan, paratophan) have a remarkable effect on the excretion of endogenous and exogenous uric acid. Atophan probably promotes the formation (mobilization) as well as the excretion of uric acid. Brugsch (*l. c.*) advises that it be given for two days, 7½ grains (0.5 Gm.) four times a day, to 15 grains (1 Gm.) three times a day, and then be suspended for a week, two weeks, or a month. It should not be used in acute gout until the attack is subsiding. Its chief use is in the treatment of chronic gout, and as a prophylactic against recurring acute attacks.

Alkalies and alkaline salts (potassium, lithium, calcium and sodium carbonates, or bicarbonates, and citrates) enjoy

a certain reputation, given either independently or as adjuvants to the above remedies. The administration of these substances should be suspended when the urine becomes amphoteric or alkaline. Potassium is the base usually preferred, while sodium has been considered injurious. Lithium has no particular virtue in gout. Iodids are of some value in chronic "gouty" and "rheumatic" conditions, although their action is not clearly understood.

Piperazin, lycetol, lysidin, quinic acid, sidonal, citarin, urosin and similar preparations have been recommended from time to time because of their supposed power of dissolving or increasing the solubility of uric acid, and thus hastening its elimination. Piperazin was used very extensively at one time, but neither this drug nor its successors have found any permanent place in practical medicine. Piperazin, like lithium, dissolves uric acid in the test-tube, but is of little or no value in the body.

TREATMENT OF THE ACUTE ATTACK.

The patient with an acute attack of gout, associated with fever, should be confined to bed. Usually on account of the painfulness of the affected joint or joints, he will have no desire to leave it. The temperature should be taken at least three times a day, and a liquid, preferably a milk diet, prescribed. Broths are not advisable. Water should be given freely, either plain or in the form of the "Imperial Drink."* The affected joints should be protected from pressure by a cradle or splint. In the case of the foot a large pad should be placed beneath the tendo achillis to prevent pressure upon the heel. The joints may be wrapped in raw cotton, or may be surrounded with gauze, which has been soaked with a saturated solution of sulphate of magnesia. Many patients, however, prefer the old-fashioned, though "unscientific" lead-water and laudanum, tr. opii, 2 fluidounces (60 mils); liq. plumbi subacetatis, 1 fluidounce (30 mils); aquæ q. s., 16 fluidounces (500 mils), applied on lint and covered with oiled

* Imperial Drink: 1 to 2 teaspoonfuls of cream of tartar, dissolved in hot water or barley-water, to be flavored with lemon-peel and slightly sweetened. Drink freely when cool.

silk or waxed paper. In general, warm applications are preferable to cold. The patient's bowels should be opened by a dose of calomel, 2 to 4 grains (0.13 to 0.26 Gm.), followed up by sulphate of magnesia the following morning. If desired, the time-honored massa hydrargyri ("blue pill") may be used instead of calomel. Subsequently the bowels should be freely opened from time to time by saline laxatives. Potassium bicarbonate or potassium citrate in 15- or 30- grain (1 or 2 Gms.) doses should be given every three hours, freely diluted with water, in order to promote elimination. If the urine becomes alkaline, the dose should be reduced. Colchicum in the form of the tincture of colchicum-seed may be given at similar intervals in 15-minim (1 mil) doses; the dose should be gradually reduced after an effect is obtained.

If colchicum causes vomiting or diarrhea, or does not relieve the pain, some preparation of salicylic acid may be employed, as in the treatment of rheumatism. Aspirin and salicin are sometimes to be preferred to sodium salicylate because they are far less irritating to the stomach. The dose of salicin is double that of sodium salicylate.

Atophan, 7½ grains (0.5 Gm.), four times a day, may be used before the attack, if premonitory symptoms are present, or after the acute stage has passed. It should be continued for two or three days, stopped, and then repeated at weekly intervals.

After a few days the fever usually falls, but the arthritic symptoms may persist for a week or two. With the subsidence of the acute symptoms, the diet may be increased by the addition at first of toast and cereals, and later of other articles given in the purin-free diet (*v. s.*). With complete convalescence, a diet with a moderate amount of purin and protein food is permissible. So long as the joints remain tender, local applications will suffice. Subsequently gentle massage, passive movements, and douching with alternate hot and cold water will be useful to restore function. Bier's treatment by passive hyperemia may be employed with good effect in some cases. Baking (superheated air) is used to favor absorption in cases in which there is persistent thickening. Potassium iodid may also be prescribed for the same purpose, in doses of 5 to 10 grains (0.3 to 0.6 Gm.), three or

four times a day. Between the attacks, prophylactic treatment should be directed against the underlying gouty diathesis.

The treatment of chronic gout and the gouty diathesis will consist almost wholly in the proper readjustment of the patient's habits of life. A careful study should be made of his previous diet, habits as to alcoholic beverages, amount and character of exercise, and like details. Special attention should also be given to the digestive, circulatory and renal functions. Thus, a vegetarian diet, while theoretically preferable to spare the uric acid metabolism, may unduly overtax the digestion. The general principles which should regulate the diet and exercise have already been explained. In these cases an occasional period of treatment at some watering place will prove useful. Gouty deposits, either about the joints or elsewhere, will be but slightly influenced by local or general treatment, but local massage, douching, packing, passive hyperemia, and hot-air treatment will be useful to improve the functions of the joints, just as in the convalescent stage of the acute cases. The thorough elimination brought about by the free consumption of water and by free sweating is also useful for the renal complications, and the various abarticular manifestations such as neuritis.

TREATMENT OF IRREGULAR GOUT AND COMPLICATIONS.

It would serve no useful purpose to describe the treatment of all the affections (many of which have already been enumerated) supposed to have a gouty basis. Where there is good reason to suspect such an underlying factor, mercurials and alkaline diuretics should be prescribed as an aid to elimination, and the usual dietetic restrictions should be instituted. Potassium iodid and guaiac resin, 5 grains (0.3 Gm.), in cachets or konseals, are valuable in many of these abarticular manifestations. In gouty sore throat Mackenzie was wont to recommend guaiac lozenges, 2 grains (0.13 Gm.). Eczema developing on a gouty basis should be treated by general measures, and by local applications. For the dry form a useful application is the following: salicylic acid, 10 to 20 grains (0.6 to 1.2 Gms.); zinc oxid and starch, each 2 drams (8 Gms.); petrolatum, ½ ounce (16 Gms.).

DIABETES MELLITUS.

Diabetes is a constitutional disease characterized by persistent disturbances of carbohydrate, and, to a less extent, of protein and fat metabolism. It is probably dependent upon pathologic changes in the pancreas. The typic clinical features are: hyperglycemia, glycosuria, polydipsia, polyuria, emaciation, acidosis and coma. According to the most recent view,[53] diabetes is the expression of a weakened function, a defect which, under proper treatment, is not essentially progressive. Glycosuria is a symptom which may occasionally be due to other causes than diabetes, but it is a safe clinical rule (Joslin) to consider it such until proved otherwise. Alimentary glycosuria ("glycosuria e saccharo") is due to the ingestion of an excess of sugar (*e.g.,* 100 to 200 grams of glucose on a fasting stomach), which cannot be "warehoused" with sufficient rapidity to prevent hyperglycemia. Renal diabetes is a condition of increased permeability* of the kidney, in which the excretory cells of that organ fail to hold back sugar even when its concentration in the blood is normal or subnormal. In true diabetes sugar is eliminated when starch is the only carbohydrate ingested ("glycosuria ex amyli"), or even when the diet is carbohydrate-free. Occasionally other sugars than glucose may appear in the urine, *e.g.,* pentose and lactose. The last mentioned may be present during lactation, and has no relation to diabetes.

Statistics seem to show conclusively that diabetes, in the last few decades, has rapidly increased in frequency in all highly civilized urban communities. The explanation, aside from greater accuracy in diagnosis, is not altogether apparent. Two factors may be important: the vastly increased consumption of sugar, and the intensity of the modern pursuit of business and pleasure. The former tends to overtax the mechanism for carbohydrate utilization; the latter may induce functional disturbances through the agency of the nervous system. Diabetes occurs at all ages, but is more common in middle life, between the ages of 40 and 60. Males are more

* This condition may be temporarily induced in man or animals by the administration of phloridzin; hence "phloridzin diabetes."

often attacked than females. Not rarely both husband and wife are affected, which, as in obesity, suggests the effect of dietetic habits. The relation of diabetes to pulmonary tuberculosis is probably accidental: their occasional coincidence is not surprising if we consider the frequency of the latter disease. Toxic and infectious changes in the islands of Langerhans are believed to be important by von Noorden, who found a history of tonsillitis in 15 per cent. of his cases.[54] Gout, obesity,* neuroses and psychoses, whether in the patient's own history or in that of his antecedents, may have a more direct connection. Occupations involving a sedentary life or mental strain and worry predispose to this disease. At times more obvious nervous insults—emotional shock, trauma, brain injuries, and the growth of brain tumors†—may bring about glycosuria. The undue frequency of diabetes among the Jews is not readily explained, but may be attributed to a combination of several of the above causes: heredity, nervous temperament, worry, diet and physical inactivity.

The generally accepted view of the pathogenesis of diabetes attributes the disturbance of carbohydrate metabolism to a deficiency in the internal secretion of the pancreas, which, in turn, has its basis in a peculiar degeneration of the cells of the islands of Langerhans (Opie, Allen and others). In the vast majority of cases enough functionating pancreatic tissue is left to afford the necessary minimal tolerance for carbohydrate, provided that the diet is properly adjusted to the weakened function. The nervous system has a profound influence upon carbohydrate metabolism, but doubtless acts through the agency of the pancreas. Many authorities believe that the sugar metabolism is regulated by a nice correlation between the sympathetic, the endocrine glands and the pancreas. Thus, nervous stimuli passing from the medulla by way of the sympathetic induce secretion of the adrenals and related structures. These internal secretions, in turn, stimulate the liver (the principal sugar "warehouse") to produce (or release) sugar. The internal secretion of the pancreas, on the

* Fifteen per cent. of the cases of alimentary obesity develop diabetes. (Kisch.)

† A diabetic patient under observation at the Philadelphia General Hospital had a stroke at the age of 40 (cerebral syphilis).

other hand, inhibits the liver, but in its turn is controlled by the internal secretions of the thyroid, hypophysis, etc. Thus, increased secretion of the thyroid, as in hyperthyroidism, tends to lessen the inhibitory action of the pancreas, and may cause glycosuria. This interesting and complicated hypothesis is of more interest in the diagnosis of obscure lesions of the ductless glands than in the diagnosis and treatment of diabetes. For practical purposes it may be disregarded.

The most frequent *pathologic lesion* in diabetes consists of a vacuolation, and eventually an atrophy of the islands of Langerhans, and, in particular, of certain so-called "beta" cells. Extensive disease of the pancreas (pancreatitis, cancer) may exist without diabetes if the "islands" are spared. Other pathologic lesions frequently found in association with diabetes are: general arteriosclerosis (important in the etiology of gangrene), pulmonary tuberculosis, cirrhosis of the liver, chronic diffuse nephritis, and tumors or other lesions of the brain.

The ordinary sugars (disaccharids) and starches (polysaccharids) of the diet in the process of digestion are converted into monosaccharids (principally glucose), and absorbed as such. The glucose is carried in the portal blood to the liver, and subsequently through the general circulation to the muscles, and is stored in these localities as glycogen. Under normal conditions the amount of glucose in the blood never exceeds 0.14 per cent. (usually 0.1 per cent.), and only traces —too small to be detected by ordinary clinical tests—are excreted in the urine. However, if very large amounts of sugar are ingested, *e.g.,* more than 100 grams of glucose, the sugar content of the blood may rise slightly, and the kidneys may allow sugar to escape, even in normal persons. Starch, being slowly absorbed, is always safely "stored" by the normal individual. After the glycogen has been deposited, it is held in reserve, and released in small amount as required for the production of heat and energy. In diabetes glucose is either not stored or is mobilized in excessive amounts. In mild cases a portion may be utilized ("burnt"); in more severe cases all, or nearly all, is lost in the urine, and yields no heat or energy to the organism. (The loss of energy from this cause is often very great. Thus, a very severe case may

eliminate as much as 12.5 liters (quarts) of urine containing 750 grams (6 per cent.) of glucose. This corresponds to 3000 calories, sufficient for the daily maintenance of an adult male of average weight.) In either case the sugar content of the blood is increased (hyperglycemia), ranging from 0.14 per cent., the extreme upper limit of the normal, to 0.5 per cent. or more. As a result the kidney is unable to hold the sugar back, and glycosuria ensues. In the mildest forms of diabetes elimination of sugars from the diet may suffice to banish glycosuria, but ordinarily restriction of starches is also essential. In severe cases the proscription of carbohydrates is insufficient, and protein must also be limited, since sugar may be derived from protein, and possibly even from fat. Different proteins are capable of yielding varying amounts of glucose (100 grams of isolated proteins may yield from 48 to 80 per cent. of sugar, according to Janney[55]), depending upon the amino-acids which they contain. When protein is being converted into sugar, and excreted as such, the proportion of glucose (dextrose) to total nitrogen in the urine rises. This is known as the D : N ratio. When all available glucose is being wasted, as in severe diabetes during fasting, this may be as high as 3.65 to 1 (Lusk,*) or 3.65 to 6.25, since 1 gram of N is equivalent to 6.25 grams of protein. In diabetes the respiratory quotient (see p. 457) is often low, because protein is being burned instead of carbohydrate. In severe cases abnormalities in the metabolism of fats are also observed. If carbohydrates are not being burned, the economy, for some reason not altogether clear, is unable completely to oxidize fats, and intermediary products of fat metabolism appear in the blood and urine.† Beta-oxybutyric acid is the primary substance formed, and from it the diacetic acid and acetone of the urine are derived. When the accumulation of these substances in the body is large we have the condition known as acid intoxication, which is manifested by typic clinical symptoms, and by various laboratory findings. The latter comprise diminution of the carbon dioxid tension of the alveolar air[56] and of the venous blood,[57] increase of

* 3.40 to 1 (Janney).

† The expression "fats burn in the fire of the carbohydrates" (Naunyn) is a vivid though not very illuminating statement of this fact.

the ammonia excretion in the urine, and excessive excretion of beta-oxybutyric acid and its derivatives in the urine. These estimations either require special apparatus or demand some technical skill. On the other hand, the simple qualitative tests for diacetic acid (ferric chlorid) and acetone in the urine are simple in the extreme. Allen finds that the latter test is easily applied to the blood-serum, and is valuable in the prompt diagnosis of acidosis. According to Henderson,[58] a practical guide to the degree of existing acidosis lies in the determination of the daily amount of sodium bicarbonate, which must be administered in order to render the urine amphoteric. Normally this should not require over 10 grams. In acidosis very large amounts are necessary—frequently more than it is wise to administer by the mouth (Joslin does not favor the use of sodium bicarbonate as a routine treatment of acidosis).

The ordinary tests for sugar in the urine, as well as the qualitative tests for acetone and diacetic acid, are so well known that it would be needless to describe them. The copper tests are universally used on account of their convenience, but in doubtful cases it is wise to confirm them by the simple but reliable fermentation method. Recently Benedict has modified Fehling's test, making it more delicate and more reliable. Solutions of slightly different composition are employed for the quantitative and qualitative estimations. The qualitative test will be described later, since with the Allen treatment it is customary to entrust the frequent analyses to the patients themselves.

Diabetes is practically always a chronic affection, but the presence of the disease may long be overlooked, and only be revealed by the acute onset of some serious, or even fatal, complication. In children the disease pursues a rapid and usually fatal course, but Riesman has recently described mild cases occurring at this period of life.[59] In young adults the most typical cases of the disease with the classical symptoms —excessive thirst, polyuria and emaciation—are observed. In persons of middle age the disease may be associated with obesity or gout, and pursue a relatively mild course. Diabetes in the aged is also marked by a paucity of symptoms, so much so that many aged persons will not tolerate any systematic

treatment. Nevertheless at this period of life there is a special tendency to vascular and pulmonary complications.

The onset of the disease, though rarely acute (*e.g.,* following nervous shock), is usually insidious. The patient's attention may be drawn to its existence by the undue frequency of urination,* itching of the skin, and rapid emaciation, or by the onset of complications such as boils, gangrene, failing vision and coma. I have a patient under observation at present, who did not notice any deviation from her usual health until the onset of gangrene of the toes. In many instances the physician discovers the presence of the disease in the course of routine investigation of the urine, as when examining for life insurance. From a therapeutic standpoint diabetics may be divided into the mild, the moderately severe and the severe, divisions which are based on tolerance tests to be described in a subsequent paragraph.

The course of a typical case in a young adult is marked by the passage of large quantities of pale urine (sometimes more than ten times the normal amount) containing varying percentages of glucose, by excessive thirst and appetite, and by progressive emaciation. Less important symptoms are dryness of the skin, itching, flatulence, flushing of the cheeks, and a red, denuded tongue. In some patients the contrast between the wasted chest and the distended abdomen is striking. The duration of these cases is on the average less than three years. (The figures of Joslin[60] show that 121 fatal cases under 30 years of age had an average duration of 2.7 years). In patients over 30 the average duration was more than twice as great.

Diabetics seldom die from the simple progress of the disease itself. They either fall a prey to acid intoxication (nearly two-thirds of the cases), or to some associated or complicating acute or chronic disease. The most common complicating diseases are: cardiovascular and renal affections, cancer, pulmonary tuberculosis, pneumonia and septic conditions, like gangrene and erysipelas.

The advent of *acid intoxication,* which, as mentioned, carries off approximately two-thirds of our patients, may be

* If this is absent one may speak of "diabetes decipiens."

gradual, with frank warning symptoms, or it may be quite unheralded. The detection in the urine of acetone, diacetic acid, and increasing amounts of ammonia may forewarn us of danger in season to permit the institution of preventive measures (administration of sodium bicarbonate, fasting). Clinically, malaise, vertigo, drowsiness, and epigastric distress may precede the onset of the typical dyspnea, stupor and coma. (Joslin[61] says that the occurrence of the following symptoms demand investigation: "anorexia, nausea, vomiting, restlessness, unusual fatigue, excitement, vertigo, tinnitus aurium, drowsiness, listlessness, discomfort, painful or deep breathing.") The coma is differentiated from that of uremia by examination of the urine, and by the peculiar deep, rapid, but regular breathing ("air hunger"). This differs entirely from Cheyne-Stokes breathing, as well as from Biot's respiration, both of which, however unlike each other, are characterized by intervals of apnea. Some persons with a well-developed sense of smell can distinguish the "fruity" odor of acetone from the urinous odor of uremia. The importance of the determination of the CO_2 tension of the alveolar air* and of the blood has been referred to above. After diabetic coma is well developed the patient seldom recovers; this is in striking contrast to uremia, in which active therapeutic interference is often successful.

Some of the minor symptoms, as well as the complications of diabetes, are classified, and in certain instances briefly characterized in the paragraphs which follow.

The mental state of diabetics tends toward depression and moroseness, which in occasional cases may amount to melancholia. Irritability, restlessness, and impatience under dietetic restraint are also said to be characteristic. Some patients indulge secretly in carbohydrate food in spite of the strictest injunctions to the contrary. When the patients are intrusted with the examination of the urine, and to a certain extent with the regulation of their own diet, there is less difficulty on this score. Peripheral neuritis is a common complication, and is accompanied by the usual signs: pain, numbness, and

* A simple instrument for the estimation of the CO_2 tension of the alveolar air is now on the market. (Hynson and Westcott.)

loss of reflexes. In some cases tabes is simulated, on account of the coexistence of optic atrophy and bilateral neuritis of the lower extremities. Diabetes is one of the conditions which may cause bilateral sciatica. Disturbances of vision and of hearing are frequent symptoms or complications of this malady. According to Saundby[62] the following are the most important causes of diabetic failure of vision: weakness of the muscles of accommodation, amblyopia and cataract.

There are no very distinctive changes in the blood in diabetes aside from the increased percentage of sugar and the presence of an undue amount of emulsified fat (lipemia). In the serum the latter may rise like cream. The cause and significance of the latter phenomenon is not understood (Bloor).

Diabetes does not ordinarily lead to endocarditis, and only rarely leads to pericarditis, but fatty heart, hypertrophy and dilatation of the heart, and vascular sclerosis are common incidents of the disease which give rise to serious symptoms: dyspnea, anginoid pains, gangrene, and sudden death.

Pneumonia is the most frequent respiratory complication of diabetes. It may be either croupous or catarrhal in type, and may terminate, more frequently than in normal persons, in gangrene or in abscess. Other pulmonary complications are emphysema, edema, infarct and pleurisy.

Ordinarily the mouth is dry, the tongue and lips are red, and the gums inflamed. Pyorrhea, dental caries, and toothache are frequently encountered. The large quantities of water and of coarse food often ingested lead to dilatation of the stomach and catarrhal conditions of the stomach and bowels, accompanied by dyspeptic symptoms, and by constipation or diarrhea. In rare instances the clinical picture of diabetes is characterized by pigmentation of the skin and symptoms of cirrhosis of the liver—hence the term "bronzed diabetes."

Chronic diffuse nephritis is a frequent complication, but hyaline changes in the kidney of less serious import are a common cause of albuminuria. Acute nephritis is very rare (3 cases in 10,000—von Noorden). The irritating quality of the urine causes pruritus vulvæ in women and balanitis in men. Women are frequently sterile, or if they become pregnant are likely to abort. Where abortion has not occurred

spontaneously its induction has been urged as a life-saving measure for the mother. With the starvation treatment Joslin[63] believes that pregnancy can often be carried through to a successful termination. In men diabetes may cause impotence.

Rapid wasting of the subcutaneous fat, in spite of the ingestion of enormous amounts of food, is one of the striking features of severe diabetes. Complications affecting the skin and its appendages are peculiarly common. Eczema, dermatitis, pruritis, pigmentation, xanthoma, purpura, boils, carbuncles, ulceration and gangrene are all seen with more or less frequency. The nails may be brittle, the hair harsh and scanty, and the skin dry. Occasionally, on the other hand, profuse sweats are encountered. Recently purulent arthritis of the knee was observed in a case of diabetes.

TREATMENT.

Whether it concern prophylaxis, palliation or cure, the treatment of diabetes is practically synonymous with its dietetic management. Treatment by drugs has retreated more and more into the background, and, in the most recent monograph on the subject, even the use of sodium bicarbonate is discouraged. The classic dietetic *régime* has been perfected by a long line of able clinicians, so that it is now possible to prescribe a diet adjusted to the tolerance of the patient with a considerable degree of ease and accuracy. Although our ideas in reference to diet have undergone radical changes in the last three years, it will, nevertheless, be advisable to outline the treatment hitherto in use, and then to consider the innovations introduced by Allen and others. We are indebted to Janeway and others for presenting the classic method of treatment in a form which is convenient, and at the same time adapted to American dietetic habits. The aim of this form of treatment is to adapt the diet, primarily the sugars and starches, and secondarily the proteins, to the patient's tolerance, and at the same time to meet, if possible, his full nutritive requirements, according to the usual caloric standards. In cases in which it is not possible to rid the urine of sugar, additional food must be given to cover the caloric losses. Cases are classified as mild, moderately severe and severe.

The mild and moderately severe cases, if put on a carbohydrate-free diet (not more than 15 grams), containing a liberal proportion of protein and fat, promptly lose their glycosuria. The moderately severe cases will not tolerate any considerable addition of carbohydrates—not more than 1 ounce (30 Gms.) in the twenty-four hours. In the mild cases 30 grams or more may be taken without causing glycosuria. Janeway[64] recommends that carbohydrates should be added in the form of ordinary bread, each increment consisting of 1 ounce or 30 grams (equal to 15 Gms. of carbohydrate). Bread is much more acceptable to the patient than any other form of starch, so it is advisable to satisfy this natural craving so far as possible. In order to permit variety Janeway has prepared a table of "equivalents," showing the quantity of various carbohydrate foods which correspond to 1 ounce of bread. Thus the patient is permitted to substitute 10 ounces (296 mils) of milk, or 5 ounces (127 Gms.) of boiled oatmeal for each ounce (30 Gms.) of bread allowed (see Table IV, p. 527). In any given case carbohydrate is gradually increased till sugar reappears in the urine. The patient is then put on a strict diet for a day or two, and thereafter is permitted to take two-thirds or three-fourths of his ascertained tolerance.

In the severe cases the patient does not become sugar-free when placed on a strict diabetic diet. The urine continues to show sugar in considerable amounts, and very often diacetic acid and acetone. In severe cases it is the rule to reduce the carbohydrate gradually, as sudden reduction is liable to increase the acidosis. In this type of case sugar is formed from protein, and it is, therefore, important to restrict the protein to a moderate extent as well as the carbohydrate.

"Hunger days" (Naunyn) and "green days" (von Noorden) are often introduced at intervals of a week or less to aid in controlling the glycosuria. A considerable percentage of the severe cases cannot be rendered sugar-free by any of these methods, and are in danger of acid intoxication if the starches are strictly limited. For their control various plans of treatment have been introduced, which have one feature in common. This is the administration of carbohydrate in some form other than that to which the patient is accustomed. These

special "cures" include milk, rice, potato, oatmeal and raw starch[65] *régimes.* The oatmeal diet, introduced by von Noorden, has had the most extensive vogue. It was at one time supposed that oatmeal starch had some specific difference which distinguished it from other starches. It is now recognized that the beneficial effects of this diet are attributable to its small protein content, and to the presence of a sufficient amount of starch to prevent the development of severe acidosis. Very frequently this diet suffices to control acidosis, and to improve the patient's condition temporarily, but the glycosuria may be aggravated. In many cases with acidosis sodium bicarbonate is added to aid in neutralizing the acidity, ½ ounce (15 Gms.) a day.

The following are taken (with unimportant modifications) from Janeway (*l. c.*). They illustrate the points that have been made, and will be found useful in arranging any diabetic diet:

I. GENERAL DIET LIST.

Eat no sugar or made dishes containing sugar; no starchy foods, such as bread of any kind, cereals, potato, rice, peas, dried beans or macaroni, soups or sauces thickened with flour, and no milk.

Try to eat every day much butter, bacon, oil on salad, and cheese, especially cream cheese.

A small amount of cream (up to 4 ounces a day) may be taken in coffee, tea or cracked cocoa (Joslin). Saccharin, ½ grain, may be used for sweetening.

Whisky with water, a light Rhine wine or claret may be drunk with dinner and supper (if specially ordered).

Foods Allowed. Clear meat soups; all meats except liver; eggs in any form; all fish except oysters, clams and scallops.

As desserts, jellies or custards, or ice-cream made with cream and eggs, sweetened with saccharin, and flavored with vanilla, coffee or brandy.

Cream, cheese and vegetables from Joslin's 5 per cent. list (see Table VII).

Mild cases are allowed, in addition, vegetables and fruits from the 10 per cent. list (Joslin), 3 ounces daily.

II. STANDARD STRICT DIET.
(Janeway—Modified).

Breakfast. Coffee with 1½ ounces cream; 2 eggs cooked with ½ ounce butter; 3 ounces ham.

Luncheon. Bouillon with 1 raw egg; 3 ounces sirloin steak, chicken or leg of lamb; 1 ounce bacon.

Vegetables from 5 per cent. list, 2 tablespoonfuls, with ½ ounce butter.

Dessert made with 1 egg and 1½ ounces cream.

Six ounces wine, or 1 ounce whisky or brandy.

(The following recipes for desserts are suggested: Baked custard: One egg, 1½ ounces of cream, 2½ ounces of water; 2 or 3 ½-grain saccharin tablets, 8 drops of vanilla essence. Beat up well, pour into a buttered dish, grate a little nutmeg on top, and bake twenty minutes. Coffee ice-cream: 1½ ounces of cream, 1½ ounces of water; 1 ounce of strong coffee, 2 or 3 ½-grain saccharin tablets. Dissolve, add 1 egg, well beaten. Mix in a saucepan and beat slowly with stirring until it thickens. Set aside until cool, then freeze).

Afternoon, tea with ½ ounce cream.

Dinner. Any clear soup; 3 ounces fish (salmon, shad or mackerel), with ½ ounce butter; ¼ pound roast pork, beef, mutton, turkey or lamb-chops.

Vegetables from 5 per cent. list, 2 tablespoonfuls, with ½ ounce butter.

Salad with ½ ounce oil in dressing; 1 ounce cheese (English, pineapple, Swiss or full cream); 6 ounces wine, or 1 ounce whisky or brandy. Demitasse of coffee.

Protein	126 grams;	504 calories.
Fat	222 grams;	1998 calories.
Carbohydrate	15 grams;	60 calories.
Alcohol	30 grams;	210 calories.
		2772 calories.

III. STANDARD DIET WITH RESTRICTED PROTEIN.
(Janeway—Modified).

Breakfast. Coffee with 1½ ounces cream; 2 eggs with ½ ounce butter; 1 ounce bacon.

Luncheon. Two eggs; 1 ounce bacon; 2 ounces lamb-chops (1), ham (2), beefsteak (3), chicken (4), or fish (5), broiled

with ½ ounce butter. (Each day select meat with same number for luncheon and dinner.)

Vegetable from 5 per cent. list, 2 tablespoonfuls, with ½ ounce butter.

Dessert made with 1 egg, 1½ ounces cream.

Six ounces wine, or 1 ounce whisky or brandy.

Afternoon, tea with ½ ounce cream.

Dinner. Any clear soup; ¼ pound roast pork (5), beef (4), mutton (3), turkey (2), chicken (1), or lamb (1). (Each day select meat with same number for luncheon and dinner.)

Vegetables from 5 per cent. list, 2 tablespoonfuls, with ½ ounce butter.

Salad with ½ ounce oil in dressing; 1 ounce cream cheese; 6 ounces wine, or 1 ounce whisky or brandy. Demitasse of coffee.

Protein	82 grams;	328 calories.
Fat	215 grams;	1935 calories.
Carbohydrate	15 grams;	60 calories.
Alcohol	30 grams;	210 calories.
		2533 calories.

IV. TABLE OF EQUIVALENTS.

(Janeway).

Breads.	Per cent. carbohydrates.	Amt. in ounces equal to 1 ounce (30 Gms.) of white bread.
White	51-55	1
All other	47-53	1
Rolls and biscuit	52-60	1
Cornbread	46	1-⅛
Crackers, average	69-72	¾
Cereals.		
Oatmeal, boiled	11.3	5
Hominy, boiled	17.8	3
Macaroni, boiled	15.8	3-¼
Rice, boiled	25.4	2-⅛
Tubers and Legumes.		
Potatoes, cooked	18-20	3
Parsnips	13	4
Beans, baked	20	2-¾
Beans, lima, cooked	20	2-¾
Peas, green, cooked	15	3-½

	Per cent. carbohydrates.	Amt. in ounces equal to 1 ounce (30 Gms.) of white bread.
Milk.	4-5	10
Fruits.		
Apples, apricots, and pears	12-14	4
Cherries	15	3-½
Huckleberries	16	3-¼
Plums	20	2-½
Bananas	22	2-½
Nuts.		
Filberts	12	4-½
Almonds	15	3-½
Peanuts	22	2-½

Additional allowances for mild cases: List of vegetables and fruits with less than 12 per cent. carbohydrates; 3 ounces of any one of these may be taken daily.

Vegetables 5 to 10 per cent:: Onion, squash, turnips, okra, carrots.

Fruits below 12 per cent.: Lemons, watermelons, strawberries, gooseberries, muskmelons, cranberries, blackberries, currants, grapefruit, oranges, raspberries, sour apples.

Nuts: Butternuts, hickorynuts, walnuts.

V. GREEN DAYS.
(von Noorden—Janeway).

Breakfast. One egg, boiled or poached; cup of black coffee.

Dinner. Spinach with a hard-boiled egg; ½ ounce bacon; salad, with ½ ounce oil; 6 ounces of wine or 1 ounce of whisky or brandy.

4.30 P.M. Cup of beef-tea or chicken-broth.

Supper. One egg, scrambled, with tomato and a little butter; ½ ounce bacon. Cabbage, sauerkraut, string-beans or asparagus. Cup of tea.

One-half ounce of sodium bicarbonate in the twenty-four hours.

The treatment by prolonged fasting was popularized in France by Guelpa,[66] who employed it in connection with free purgation, not only for diabetes, but also for other chronic conditions, all of which he attributed to "autointoxication." In the intervals between fasts—he usually repeated them several times—he employed a milk or a strict carbohydrate-free

diet. He did not think it was necessary to keep the urine constantly free from sugar in the early stages of treatment. He and his followers obtained brilliant results in many cases. Dr. Allen,[67] on the basis of careful animal experiments, adopted Guelpa's method of prolonged fasting, and followed it up by a very strict diet, consisting principally of green vegetables. He first tested the carbohydrate tolerance by giving increasing amounts of green vegetables, etc., and then in succession the protein, and, if necessary, the fat tolerance. If during the tolerance tests sugar reappeared, he introduced a single fast day, and reduced the particular foodstuffs by a safe margin. He aimed to give 1 gram of protein for each kilogram of body weight, and enough fat to maintain the nutrition, if this were easily possible. Unlike the classic treatment, the starvation treatment does not make a point of maintaining or increasing the body weight. If the patient is obese it is even desirable to reduce the weight considerably, as this facilitates the maintenance of tolerance. The diabetic is kept continuously sugar-free, and acidosis is controlled by weekly fasts. Since no attempt is made to supply a definite number of calories, the administration of extensive amounts of fat is rendered unnecessary. In fact, fats are increased with caution, as the addition of butter or olive oil may cause glycosuria or ketonuria. Allen[68] bases his treatment not on any hypothesis of autointoxication, but on the theory that a function, if impaired, may be strengthened by rest. Under this treatment the carbohydrate metabolism usually improves, so that in time the patient's tolerance is materially increased. For this reason the tolerance should be tested and retested at long intervals, and the diet readjusted in relation to the new findings. In order to carry out this rather elaborate treatment, it is necessary for the urine to be frequently examined. Both Allen and Joslin believe that it is better for the patient to make these tests himself, which he may readily do by Benedict's qualitative method.

VI.

Benedict's solution is permanent, and at the same time ten times as sensitive to sugar as is Fehling's solution. Chloroform, uric acid, and creatinin do not interfere with the test:

Copper sulphate (pure crystallized)	17.3
Sodium citrate	173.0
Sodium carbonate (crystallized)	200.0
Distilled water	ad 1000.0

(The citrate and carbonate are dissolved in about 700 mils of water with the aid of heat, and the mixture poured into a large beaker. The copper is dissolved in about 100 mils of water, and poured slowly and with constant stirring into the first solution. The mixture is then cooled and diluted to 1 liter—quart.)

For the detection of glucose in urine, 5 mils of the reagent are placed in a test-tube, and 8 to 10 drops (not more) of urine added. Heat to vigorous boiling for one or two minutes, and allow to cool. In the presence of glucose the entire body of the solution will be filled with a precipitate, which may be of a red, yellow or greenish tinge. If the quantity of glucose be under 0.3 per cent. the precipitate forms only on cooling. If no sugar be present, the solution remains clear or shows a faint-blue turbidity. It is often more convenient to place the test-tubes in a beaker of boiling water, where they should remain for five minutes. If sugar reappears, the patient is instantly to take a fast day in bed, and then to reduce the diet somewhat below the previous limit. Additional fast days are advised every week or two, and the patient is weighed weekly to avoid increase in weight.[69]

SUMMARY OF DIETETIC TREATMENT.[70]

"*Preparation for Fasting.*—In very severe, long-standing, complicated obese, and elderly cases, as well as in all cases with acidosis, without otherwise changing habits or diet, omit fat, after two days omit the protein, and then halve the carbohydrates daily until the patient is taking only 10 grams; then fast. In other cases begin fasting at once.

"'*Fasting.*—Fast four days, unless sugar-free earlier. Allow water freely; tea, coffee, and clear meat broths as desired.

"*Intermittent Fasting.*—If glycosuria persists at the end of four days, give 1 gram protein and ½ gram carbohydrate per kilogram body weight for two days, and then fast again for three days unless earlier sugar-free. If glycosuria remains, give the protein as above, but with no carbohydrate for three days, and then fast for one or two days as necessary. If there is still sugar, give protein as before for four days, then fast one, and then gradually increase the periods of feeding, one day each time, until fasting one day each week. Uncomplicated cases rarely fail to become sugar-free by this method.

"Carbohydrate Tolerance.—When the twenty-four-hour urine is free from sugar, add 150 grams of 5 per cent. vegetables (3 per cent. available carbohydrate). The approximate content of carbohydrate would be 5 grams, and not 7.5 grams, due to the presence of cellulose and the lower percentage of starch in such vegetables as lettuce. Continue to add 5 grams carbohydrate daily in this form up to 20 grams, and then add 5 grams every other day in the form desired until glycosuria appears.

"Protein Tolerance.—When the urine has been sugar-free for two days, add about 20 grams protein (3 eggs) and thereafter 15 grams protein daily in the form of meat until the patient is receiving at least 1 gram protein per kilogram body weight, or, if carbohydrate tolerance is zero, only ¾ gram per kilogram body weight.

"Fat Tolerance.—While testing the protein a small quantity of fat is included in the eggs and meat given. Add no more fat until the protein reaches 1 gram per kilogram body weight (unless the protein tolerance is below this figure), but then add 5 to 25 grams daily, according to previous acidosis, until the patient ceases to lose weight or receives about 30 to 40 calories per kilogram body weight.

"Reappearance of Sugar.—The return of sugar demands fasting for twenty-four hours, or until sugar-free. Resume the former diet, except that the carbohydrate is diminished, one-half until the urine has been sugar-free for one month, and it should not then be increased more than 5 grams a month.

"Weekly Fast Days.—Whenever the tolerance is less than 20 grams carbohydrate, fasting should be practised one day in seven; when the tolerance is between 20 to 50 grams carbohydrate, upon the weekly fast day 5 per cent. vegetables and one-half the usual quantity of protein and fat are allowed; when the tolerance is between 50 and 100 grams carbohydrate, the 10 and 15 per cent. vegetables are added as well. If the tolerance is more than 100 grams carbohydrate, upon weekly fast days the carbohydrate should be halved."

The following tables are taken from the cards issued by Joslin;* these cards, on one side, give an outline of the treatment similar to that just quoted, and, on the other, furnish the necessary data for arranging the diet:

VII.

Strict diet, meats, fish, broths, gelatin, eggs, butter, olive oil, coffee, tea, and cracked cocoa.

Foods arranged approximately according to per cent. of carbohydrates.

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* For sale by Thos. Groom and Company, 105 State Street, Boston, Mass.

Vegetables (fresh or canned):

5 per cent.*		10 per cent.	15 per cent.	20 per cent.
Lettuce	Tomatoes	Pumpkin	Green peas	Potatoes
Cucumbers	Brussels sprouts	Turnips	Artichokes	Shell beans
Spinach	Water-cress	Kohl-Rabi	Parsnips	Baked beans
Asparagus	Sea-kale	Squash	Canned lima beans	Green corn
Rhubarb	Okra	Beets		Boiled rice
Endive	Cauliflower	Carrots		Boiled macaroni
Marrow	Egg-plant	Onions		
Sorrel	Cabbage	Mushrooms		
Sauerkraut	Radishes			
Beet-greens	Leeks			
Dandelion-greens	String-beans			
Swiss chard	Brocali			
Celery				

* Reckon available carbohydrates in vegetables of 5 per cent. group as 3 per cent.; of 10 per cent. group as 6 per cent.

Fruits:

5 per cent.	10 per cent.	15 per cent.	20 per cent.
Ripe olives (20 per cent. fat)	Lemons	Apples	Plums
Grapefruit	Oranges	Pears	Bananas
	Cranberries	Apricots	Prunes
	Strawberries	Blueberries	
	Blackberries	Cherries	
	Gooseberries	Currants	
	Peaches	Raspberries	
	Pineapple	Huckleberries	
	Watermelon		

Nuts:

5 per cent.	10 per cent.	15 per cent.	20 per cent.
Butternuts	Brazil nuts	Almonds	Peanuts
Pignolias	Black walnuts	Walnuts (Eng.)	Chestnuts (40 per cent.)
	Hickory	Beechnuts	
	Pecans	Pistachios	
	Filberts	Pinenuts	

Miscellaneous (5 per cent.):

Unsweetened and unspiced pickles, clams, oysters, scallops, liver, fish-roe.

(30 grams = 1 ounce) Contain approximately	P. Gms.	F. Gms.	C. Gms.	Cal.
Oatmeal, dry wgt.	5	2	20	120
Meat (uncooked, lean)	6	3	0	50
Meat (cooked, lean)	8	5	0	75
Broth	0.7	0	0	3
Potato	1	0	6	25
Bacon	5	15	0	155
Cream, 40 per cent.	1	12	1	120
Cream, 20 per cent.	1	6	1	60
Milk	1	1	1.5	20
Bread	3	0	18	90
Butter	0	25	0	225
Egg (one)	6	6	0	75
Brazil nuts	5	20	2	210
Small orange or ½ grapefruit.	0	0	10	40
Vegetables, 5 and 10 per cent. group	0.5	0	1 or 2	6 or 10
Oysters (six)	6	1	4	50

For the satisfactory measurement of diabetic diets, food scales are necessary,—at least, until the patient is thoroughly familiar with food, and particularly carbohydrate values. (The "Harvard Scale" (Troemner) is suitable for the purpose. Convenient spring scales are also made which permit of the adjustment of the scales after each addition of food, and thus obviate calculations.)

If the diet has been so adjusted that sugar is banished from the urine and acidosis controlled, it seems probable that the disease will show no necessary tendency to progress, and toleration will improve to a certain degree. Advantage may be taken of this gain to increase the diet, if toleration is retested after a period of some months. On the other hand, the impaired function, since it has a pathologic basis, is not likely to become normal again. It will be necessary, therefore, for the patient to maintain a careful diet for the balance of his life as the price of continued health.

The patient with mild diabetes may usually go about his work without interruption, provided that he adheres to the dietetic regulations. In the moderately severe and severe cases it is wise to admit the patient to a hospital, sanatorium, or nursing home until the tolerance tests have been completed, and the patient has become thoroughly accustomed to the dietetic routine. During the period of starvation, in cases

in which this method is necessary, the patient, as a rule, should be kept in bed, but an exception may be made in the case of vigorous young adults, who may be allowed to be about the room. After a patient's toleration has been determined he may be dismissed with instructions to test the urine daily, and report to the physician every week or two, depending upon the severity of the case. The reappearance of sugar demands immediate restriction of diet, and in most cases a day of fasting. It is important that the patient should lead a regular, methodic life, as free as possible from strain and excitement. Frequent warm baths are an advantage to keep the skin in good condition. It is particularly important that the genitals should be kept perfectly clean and dry, for the urine, as long as it contains sugar, is prone to cause distressing pruritus or eczema. Many of the mild cases are associated with either gout or obesity, and will be benefited by a stay at some well-regulated watering-place, where they may indulge in open-air exercise, and have the benefits of a restricted diet, mild alkaline waters, hydrotherapy and massage.

One of the newer ideas in the treatment of diabetes concerns the value of exercise. Clinical experience thus far indicates that vigorous exercise is beneficial, even in severe cases on a restricted diet. It appears to stimulate metabolism and increased sugar toleration.[71]

The drug treatment of diabetes, which once occupied a prominent place has, as I have said, been relegated to the background. (Forscheimer holds it to be still of value in the 20 per cent. of cases who will not, or can not take treatment.) This does not apply, of course, to ordinary tonic, laxative or symptomatic treatment, which may be employed on general principles as in any other chronic disease. Sodium bicarbonate is widely used as a prophylactic against acidosis, but this practice is not as popular as formerly. One-half ounce would be an average dose in the twenty-four hours. The use of sodium bicarbonate will be referred to again under the treatment of special symptoms.

Opium is the drug which has maintained its reputation best in the specific treatment of this disease. It is usually administered in the form of the crude drug, or in the form of codein. There is no question that this drug promotes the

comfort of the patient, and reduces the excretion of sugar temporarily. But its use must almost inevitably lead to the establishment of the opium habit, and with the perfection of the modern dietetic treatment its use is inexcusable, except in occasional advanced or hopeless cases, where dietetic restriction is impossible or cruel. The dosage of opium and its various derivatives naturally does not differ from the ordinary, but with prolonged use would necessarily have to be increased to obtain results..

Aside from opium, other sedatives, such as antipyrin, phenacetin, and the bromids, have enjoyed popularity in certain quarters. Thus Guelpa refers to a method of treatment employed by one of his colleagues which consisted in the alternate use of antipyrin and arsenic. By this combination both the amount of sugar and quantity of urine were diminished, but sugar was only abolished after the institution of prolonged starvation.

Arsenic was formerly a favorite remedy, but cannot be credited with any specific effects. The use of pancreatin and other products derived from the pancreas would seem to have a more rational basis, in view of the well-established pancreatic theory of the disease. Unfortunately experience does not indicate that these extracts have any specific value. In cases in which the pancreas is seriously affected, they may supplement the external secretion of this organ and improve digestion.

TREATMENT OF SPECIAL SYMPTOMS AND COMPLICATIONS.

The most important and fatal complication of diabetes is diabetic coma. The only effective treatment of this condition is prophylactic. This demands that the patient be kept under constant supervision, so that any tendency to acidosis suggested by the symptoms already mentioned, or by the presence of the ferric chlorid reaction (Gerhardt's test for diacetic acid), be immediately combated by the administration of carbohydrates and the employment of liberal doses of alkali; or, on the other hand, if we follow the newer treatment, by a sufficient period of starvation to eliminate sugar and to reduce the amount of the fatty acids. When food is

resumed the fats should be kept very low. If possible it is advisable to estimate the quantity of beta-oxybutyric acid, but it is much simpler to determine the carbondioxid tension of the alveolar air (Marriott's or Fredericia's methods), or of the blood (van Slycke's method). When first introduced the alkali therapy awakened great hopes, which have not altogether been sustained. Those who use the Allen treatment maintain that it is rarely necessary. The most effective method of administration in impending coma is by intravenous infusion. Five hundred mils of 4 per cent. sodium bicarbonate in physiologic salt solution may be slowly injected into one of the veins at the bend of the elbow, and this dose may be repeated within twelve hours, if necessary. Subcutaneous injections are unsafe on account of local irritation, or even necrosis, consequent to such procedures. Alkali therapy should be suspended when the acid reaction of the urine is abolished, but this seldom occurs. When coma is fully established, treatment is usually futile, though consciousness is occasionally restored temporarily.

Most of the complications of diabetes, such as neuritis, ocular disturbances and skin eruptions may be prevented or greatly benefited by strict dietetic treatment. Local applications or operative treatment are required for skin eruptions, boils or carbuncles, but not much benefit may be expected until the patient is rendered as sugar-free as possible. Gangrene usually occurs in the aged in whom there is coincident arterial disease. Excellent results, both in limiting the extent of the gangrene and in promoting healing after operation, have been recently credited to the starvation method. If the gangrenous areas are small, and there is no tendency to spreading they may be treated expectantly by antiseptic or aseptic methods until a line of demarcation has formed, when a conservative operation may be performed. If, however, the disease is extending rapidly, and there are evidences of extensive arterial disease, a high amputation would be indicated.

Pyorrhea and caries should be combated by careful toilet of the mouth, including cleansing with mild antiseptic solutions and painting the gums with tincture of iodine. In nephritis there will be reasons, in addition to those already mentioned, for restricting the protein,—a fact sufficiently obvious

not to require extended discussion. In tuberculosis it is a nice question between the dietetic restriction, essential in the treatment of diabetes, and the overfeeding desirable for tuberculosis. It is more important, however, to get rid of the sugar, and, coincidently, to combat the tuberculosis by rest and fresh air.

Allen, in a recent lecture at the College of Physicians in Philadelphia (November, 1916), advised bran and agar biscuits for patients on a very strict diet. These, though tasteless, serve as a good vehicle for fats, and are valuable to regulate the bowels. Joslin[72] gives the receipt for their preparation:

Bran	60 grams (2℥, 50 grs.).
Salt	¼f℥ (0.92 mils).
Agar-agar, powdered	6 grams (93 grs.).
Cold water	100 mils (3.38f℥).

"Tie the bran in cheesecloth and wash under cold-water tap until the water is clear. Bring both agar-agar and water (100 mils) to the boiling point. Add to the washed bran the salt and agar-agar solution (hot). Mold into two cakes. Place in a pan on oiled paper, and let stand half an hour; then, when firm and cool, bake in a moderately hot oven thirty to forty minutes.

"Naturally the bran muffins will be far more palatable if butter and eggs are added. This may be done provided that the patient allows for this in the diet. If the patient is not upon a measured diet, then considerable latitude can be employed in making the bran cakes."

The use of gluten breads and other diabetic foods has not been advised in the text because they are unnecessary, unpalatable to most persons, and often unreliable. Before using any particular preparation the physician should consult reliable analyses.[73]

DIABETES INSIPIDUS.

Diabetes insipidus is a rare* chronic disease characterized by the passage of excessive quantities of pale urine of

* Futcher[74] found 7 cases among 17,042 patients on Dr. Osler's service at the Johns Hopkins Hospital.

low specific gravity and free from pathologic constituents. Minute traces of albumin or sugar may be present in exceptional cases. Two varieties of the disease are described: (1) the idiopathic, which is believed to be dependent upon functional inability of the kidney to concentrate urine, and (2) the symptomatic variety. The latter is dependent upon functional or organic changes affecting the central nervous system. Functional polyuria has been observed in hysteria and following psychic causes, *e.g.*, "emotional insult." The organic variety is due to implication of the base of the brain as the result of trauma (fracture), meningitis (syphilis), or tumor (secondary carcinoma). The most frequent underlying disease is lues, which takes the form of a gummatous meningitis. Four of Futcher's 7 cases were due to this cause. According to Cushing,[75] symptomatic polyuria is dependent on either injury or disease of the posterior lobe of the pituitary, leading to increased secretory activity. He regards emotional polyuria as an expression of a neurogenic discharge of hypophyseal secretion. Disease of the floor of the fourth ventricle is also believed to be a cause of diabetes insipidus, this view being based on the celebrated experiments of Claude Bernard. The malady is more common in the young, and in those with a family history of metabolic or renal disease.

The classic symptoms are polyuria, polydipsia and polyphagia. In some cases the large appetite may be replaced by anorexia, or there may be no disturbances of the appetite. Secondary symptoms are: emaciation, dryness of the skin, diminished perspiration, distention of the bladder, constipation or diarrhea, and lumbar pain (Trousseau). On account of the lesion of the base of the brain there may be headache, ocular changes and increased reflexes. In men there may be impotence, and in women scanty menstruation or amenorrhea. In infancy diabetes insipidus may lead to enuresis (Still).

The quantity of urine may be increased to ten, or occasionally even to twenty or thirty times the normal amount. It is not uncommon for the amount of urine excreted to exceed the volume of fluid ingested. The difference is derived from the food (see chapter on Metabolism), or temporarily from the tissues. Restriction of fluid, if strictly carried out, may reduce the quantity of urine, but causes great suffering

and has no curative value. The urine is almost watery in appearance; the specific gravity usually varies between 1001 and 1003. The ordinary urinometer will suffice for clinical determinations, but slight variations cannot be accurately read.

Acute polyuria is without question a mere symptom. Chronic polyuria may also be regarded as a symptom, though if combined with other manifestations such as have been described, it forms the symptom-complex diabetes insipidus. The chief causes of chronic polyuria which have to be excluded are: diabetes mellitus and chronic diffuse nephritis. Hysteric polyuria is usually of brief duration, although recurrent attacks may occur over a long period of time. As a rule, diabetes mellitus is easily distinguished by the high specific gravity of the urine, the glycosuria, and other concomitant symptoms. The occasional cases of diabetes insipidus in which sugar appears from time to time probably bear no relation to true pancreatic diabetes, since a lesion at the base of the brain may cause polyuria, either with or without sugar. In the polyuria of chronic nephritis either albumin or casts may occasionally be missed, but repeated urinary examinations and blood-pressure estimations will usually settle the diagnosis. In the differentiation of the various forms of diabetes insipidus particular stress should be laid on the Wassermann test, and on the detection of symptoms suggestive of pituitary disease.

The prognosis of diabetes insipidus is entirely dependent on the cause. If of a functional nature it may not shorten life. Cases are mentioned lasting as long as fifty years. Many examples of the disease, however, are dependent upon serious lesions of the brain, while others are associated with acromegaly, Fröhlich's syndrome, or malignant disease of the pituitary. Several cases are on record of carcinoma of the pituitary, secondary to cancer of the breast.

TREATMENT.

Patients with this disorder, barring complications, may often follow their usual occupation, providing it is not too strenuous. A case of this sort coming under observation for the first time should be carefully studied to determine its

character and degree of severity. To do this efficiently it will be necessary to confine the patient to bed, either at home or in a hospital. Acromegaly and degeneratio-adiposo-genitalis should be excluded by a careful review of the history and detailed examination. In the latter condition there is usually an increased sugar tolerance, while in the former the opposite condition prevails. This point may be determined by the administration of 100 grams (3℥ 230 grs.) of glucose in the morning on a fasting stomach. The daily quantity of the urine should be measured, and the effect of moderate restriction of water, a salt-free diet, etc., tested. These measures are occasionally beneficial. As mentioned above, an occasional trace of albumin or sugar will not make the diagnosis of nephritis or diabetes, but it is important to use every means to exclude these conditions. A history of syphilis should be sought for, and a Wassermann test made. If headache or ocular symptoms are present, the eyes should be examined for hemianopsia, optic neuritis, etc. As these patients tend to emaciate they should be allowed a plentiful diet with an adequate supply of protein. If the appetite be excessive (polyphagia), a light lunch may be given between meals. On account of the lack of perspiration the skin is dry and harsh, and should be kept in good condition by frequent bathing, and, if necessary, by the use of emollients (cold cream and the like).

In cases in which the pituitary is incriminated, pituitrin may be administered subcutaneously, 7 to 15 minims (0.5 to 1.0 mil), twice daily, with the expectation of reducing the urinary output 50 per cent.[76] This treatment is deficient because it is not practicable to continue it more than a few days, but it may be of use during an exacerbation of the disease. Valerian is the best known palliative, and in large doses reduces the amount of the urine very considerably. Trousseau administered as much as 30 grams, and succeeded in reducing the urine from 29 to 6 liters (quarts), but induced severe gastrointestinal symptoms. Ordinarily 30 grains (2 Gms.) should be a sufficient dose. The drug has the fault of being intensely disagreeable. If moderate doses be employed, the ammoniated tincture will be found the most eligible preparation. Other sedatives have a similar effect in reducing the quantity of the urine,—opium, bromides, cannabis indica.

The same objections may be raised to the use of opium as in the case of diabetes mellitus. Nitroglycerin is credited with beneficial effects in some cases. The most generally useful drug, however, is fluidextract of ergot in doses of 15 minims (1 mil) three times a day. Here, again, prolonged use is not without danger. Belladonna does not appear to be of much value. Aside from drugs which affect the secretions directly, general tonics, such as nux vomica, iron and arsenic, are useful. If the diagnosis of syphilis be certain, "specific" treatment is in order: mercurial inunctions, potassium iodid and salvarsan. This treatment frequently relieves the headache and other general symptoms, but does not appear to have any direct effect on the diuresis.

Massage and hydrotherapy are of value to improve the general condition, while electricity (galvanic) has been applied to the base of the brain with the hope of influencing the pituitary directly.

ARTHRITIS.

The problem of arthritis in its various phases is an extremely large one, and has been the subject of a most voluminous literature. Only in the relatively recent past has it been classified with any real success, and even now authorities differ as to the best subdivisions of the types encountered. For the general purposes of treatment the subject can probably be best approached from the standpoint of the following classification, by Barker, of diseases of the joints at large.[77] This affords a very comprehensive summary of the diagnostic features of the various arthritides, and the writer has drawn freely upon it and the other works referred to:

1. The congenital arthropathies.
2. The static and the toxic degenerative arthropathies.
3. Arthropathies of circulatory origin.
4. The inflammatory arthropathies (arthritides).
5. The neuropathic arthropathies.

The first two varieties find no place in a textbook on medical treatment, although brief references will be made to the second type. The third variety, arthropathies of circulatory origin, demands brief reference because of the so-called "Bleeder's Joint"; that is to say, an articular disturbance

occasioned by hemorrhage into the joint in a patient suffering from a hemorrhagic diathesis, hemophilia, purpura or scorbutus. The treatment of this condition needs no special mention beyond the treatment of the underlying cause, except in so far as it may be influenced by the general considerations of rest, local applications, and so on. These will be included in the treatment of the other arthritides. The neuropathic arthropathies, next in the foregoing classification, also call for no particularization here. They depend for their treatment upon recognition of the underlying disturbance, and deserve mention only to direct attention to the general applicability of such measures as rest, support, the local surgical care of trophic sores and ulcers, and the like. These several varieties of arthropathies are mentioned because their symptoms, to a small degree, may resemble those of the fourth group, and differential diagnosis is at times necessary.

The inflammatory arthropathies included in the above classification are the essential subject of consideration here, and form a most important chapter in medicine.

Probably in no other department of medicine has there been more confusion in regard to nomenclature than here, and only in the very recent past has recognition of the infectious origin of many cases of arthritis brought some order out of chaos. Classification of the inflammatory arthritides is again dependent largely upon the viewpoint of the individual writer, and may be referred to a pathologico-anatomic, etiologic or clinical grouping.[78] For clinical purposes the last is the most useful of the three, and will be used here. In this classification the infectious arthritides are divided, in so far as our purposes are concerned, as follows:

1. The true chronic gouty arthritis.
2. The primary hypertrophic osteoarthritis.
3. The secondary chronic infectious arthropathies following various bacterial invasions.
4. The so-called primary chronic progressive polyarthritis; possibly a special member of the preceding group.

In connection with these four main groups, several special conditions are also to be considered:

(*a*) Chronic villous arthritis (the villous arthritis of Goldthwait).

(*b*) The chronic arthropathies of the spine.
(*c*) Still's disease.
(*d*) Heberden's nodes.
(*e*) Subcutaneous fibroid nodules.

The gouty joint will not be taken up here, as it comes properly under the general subject of "Gout" (*q. v.*).

PRIMARY HYPERTROPHIC OSTEOARTHRITIS.

Primary hypertrophic osteoarthritis is a condition which is easily recognizable, and occurs most frequently in late adult life. It is characterized, in general, by involvement of one of the larger joints of the body, such as the hip, shoulder or knee, in contrast to the more diffuse processes seen in other types; occasionally, however, more than one joint is affected. It is important that the possibility of malignant growth be not overlooked in these cases, especially when the hip is concerned, and the *x*-ray is useful in making the differentiation. The pathologic process may be unaccompanied by other obvious disturbances of health, and consists of atrophy of cartilage, roughening and hypertrophy of the bony surfaces, exostoses, lipping of the joint-margins, and so on. This condition has probably the same etiology as the types next to be described, and treatment of it can profitably be included under treatment of them.

INFECTIOUS ARTHRITIS.

It is in connection with this subvariety of arthritis that significant advances have been made in treatment. If the condition known as acute rheumatic fever, for which the salicylates are in the nature of a specific, be eliminated from consideration here, it may be said that most of the cases of febrile acute non-traumatic arthritis belong in the above category, and can be referred to a focus of infection somewhere in the body. It is even a moot question whether acute rheumatic fever should not be included here also.

The lesion may be of the smaller joints alone, or of the larger alone, though in general one or more joints of both kinds will become affected as the disease progresses or becomes chronic. At the outset the site of the arthritis may be

painful, swollen, or even inflamed to inspection, and as it becomes more chronic and spreads to other joints there intervene atrophy of the cartilage, atrophy and rarefaction of the bone, frequently overgrowth on the shaft of the bone, and thickening and contracture of the capsule and tendons. Later, as the articulations become anatomically altered, subluxation and hyperextension may occur; or, if there be much lipping and overgrowth, limitation of motion, or even complete fixation may result. There may be effusion within the capsule of the joint. At any time, or, indeed, during the entire course of the above condition, there may be slight fever and secondary anemia of more or less severity, slight leucocytosis, and enlargement of the lymph-glands. The clinical picture depends upon the degree of advancement of the disease, but when this is well established the patient becomes more or less bed- or chair- ridden, the knees, shoulders, and elbows have only limited motion, the knees are enlarged, the hands show irregular swelling of the knuckles and interphalangeal joints, the wrists may be almost ankylosed, and at any or all of these sites there may be pain upon motion, or even at rest.

In what is undoubtedly the most complete single work upon the subject of rheumatic conditions, Jones and Jones[79] have emphasized the importance of fibrositis as the underlying and characteristic pathologic phenomenon. They point out that it is the fibrous tissue which is primarily concerned in these conditions, whether the disease be seated in the muscle, nerve, cartilage or bone. They discuss the so-called "rheumatic" states under the general term "fibrositis," and the reader must be referred to this exhaustive work for a full presentation of this pathology.

In this class of cases it is generally recognized that the causative agent is an infection somewhere in the body. The sites of this infection and the kinds of organisms responsible for it are very varied.

Arthritides may also follow infectious diseases such as pneumonia, typhoid fever, meningitis, and general infections like erysipelas and puerperal infection, and one attack may suffice to render the subject permanently crippled by the time subsidence of the inflammation has occurred. However, this is not the usual picture, and in general it may be said that

patients suffering from the more or less diffuse symptoms above described carry somewhere in their bodies a source of infection, often inconspicuous, from which a slow, insidious absorption takes place. This focus may be a gonococcal infection of the genito-urinary tract, but generally a less specific type of organism is at fault. Bacteriologic studies have shown that streptococci exceed all other varieties of organism in the frequency with which they are the causative agents, and the work of Rosenow and others has shown that, of the various streptococci, the *Streptococcus viridans* and the *Streptococcus hemolyticus* are those most usually to be isolated. Experiments have indicated that strains of organisms isolated from various foci in the body may produce analogous lesions when injected into experimental animals, and that there appears to be a certain degree of selective affinity in regard to them.[80] Although the organisms mentioned seem to play the most important rôle, the staphylococcus may be equally potent, and it must not be overlooked that almost any organism may be concerned, including the possibility of infection by the various fungi and amebæ. Full consideration of this important question is impossible here, but complete presentations are available in the writings of Billings, who has been chiefly instrumental, in conjunction with his co-workers, in expanding our ideas as to what may constitute a source of infection.[81] The focal disturbance, caused by these organisms, from which the toxemia arises may be in the tonsils, accessory sinuses of the skull, teeth, gums, alveolar processes of the jaw, ears, appendix, gall-bladder, genito-urinary tract, or, indeed, anywhere in the body.

TREATMENT.

In view of what has been said, therefore, it is clear that treatment of chronic infectious arthritis depends upon the recognition of the nature and site of infection, and the steps taken to remove it or to combat it, if removal is not possible. Since discovery of the site of infection is of the first importance, it is well to consider here at some length the procedures necessary to that end.

Possibly the most important single site of absorption is to be found in the teeth, which in these conditions are often the

seat of cavities. The roots of the teeth may be extensively affected, although giving no superficial evidence of this upon mere inspection of the mouth. Obviously this condition may vary from a degree of slight decay to that in which the teeth are grossly diseased and loose in their alveolar sockets. The gums, likewise, may be greatly or slightly affected, and pressure upon them may cause the exuding of a thin pus; or they may be spongy and bloody upon slight pressure. This condition of pyorrhea alveolaris may be, in itself, sufficient to induce and to perpetuate symptoms of arthritis, and hence it demands vigorous treatment. Accumulations of tartar upon the teeth should be removed, and the gums brought into healthy condition by frequent cleansing of the mouth, the removal of tartar from the teeth, the use of weak hydrogen peroxid, astringent mouth-washes, argyrol in 50 per cent. strength, application of tincture of iodin, etc., preferably under the direction of a good dentist; notwithstanding consistent efforts, much time may be required to restore conditions to normal.

Recently Barrett and Smith[82] have described the occurrence in gums thus affected of an ameba which they describe as the *Entameba buccalis*. Basing the thought upon the results seen in other forms of amebiasis, the use of emetin hydrochlorid, the alkaloid of ipecac, has been advocated in pyorrhea, with alleged successful results in many instances. It is given subcutaneously in doses of about ½ grain (0.0324 Gm.) daily. Some few disastrous consequences[83] have followed its use in doses of 1½ grains (0.0972 Gm.) daily, and care must be exercised that it be not administered longer or in larger dosage than required. It may also be injected locally between the tooth and the gum in amounts of about 1 drop of a half of 1 per cent. solution. In any event, it should be accompanied by the measures designed to promote the hygiene of the mouth—already noted. It not infrequently happens that steps taken to rectify infectious conditions of the above nature are not complete, even when the patient has consulted a dentist, and apparently all offending members have been removed. Under these conditions the desired improvement will not take place, from which it follows that a more thorough search must be made to detect the site of trouble. To this end the *x*-ray is of great value, and it is indeed a debatable

point whether it should not be used as a routine step in every case where search is made in the teeth for a site of possible infectious absorption. *X*-rays of the teeth made in groups of two or three, by means of films placed in the mouth, will show grossly diseased processes that have escaped detection after the most thorough examinations by other means. Between the root of the tooth and its alveolar socket there may be an apparently trifling space more or less filled with carious material, merely the result of tartar. This condition may extend to the frank formation of pus which collects at the root of the tooth as in a pocket. The root of the tooth may also give further evidence of disturbance in an irregularity of its outline and rarification of its substance. The alveolar processes themselves may share in this pathology, and undergo a distinct shrinkage along the axis of the tooth.

Space is given to the consideration of these points, because of the extreme likelihood that they may be overlooked, and too much emphasis cannot be placed upon the importance of having suspicious cases studied by both dentist and röntgenologist in the most thorough manner.

In the experience of critical observers it is sometimes necessary to force the issue as to whether or not a given tooth or its socket is importantly at fault, by insisting upon removal of the tooth, even in the face of dental opinion to the contrary.

Foci in the mouth may exist, which appear trifling on their own account or from the purely dental standpoint. It is only when they are viewed from the larger viewpoint of the relation they bear to systemic conditions, however, that their true importance appears, and under these circumstances the physician may have to overcome considerable opposition. Obviously a chain is as strong as its weakest link, and since it has been well proved that otherwise relatively trifling foci can be productive of serious consequences, one cannot be sure of having done one's full duty until there remains no longer ground for the slightest suspicion at any point.

Just as the teeth may be the site of suspected trouble, so the tonsils may be similarly at fault. If they be grossly implicated, and this be evident upon simple inspection of the throat, there can be no doubt as to the steps indicated. How-

ever, in the experience of many observers, mere inspection is quite insufficient to incriminate or to absolve them, and recourse must be had to a more thorough examination at the hands of the nose and throat specialist. Even he may, at times, fail to appreciate upon examination alone the amount of diseased structure, and tonsillectomy may later show centrally located septic foci. Relatively trifling crypts may harbor necrotic material, and even the space between the reduplications of tonsillar tissue may suffice for the lodgment of food imperfectly removed or *débris,* which acts as a culture medium. The importance of such apparent trifles is sometimes to be seen not so much in the relation they themselves bear to the systemic joint disturbance as in their cumulative effect in conjunction with other apparently trifling foci elsewhere.

When the tonsils, therefore, are found to be affected it is very doubtful whether, in such clear-cut instances, any steps short of complete removal are of real avail. In a few cases, especially where there is some strong contraindication for removing them, expression of the contents of the tonsillar crypts may be of value.

In the same way the ears should be made the subject of a most critical investigation by one trained in this line, though in general the ears are less frequently at fault than are the teeth and tonsils. Examination of the head for foci of infection is not complete unless the accessory sinuses of the skull are also included. This means transillumination by the specialist and several *x*-ray pictures before the data are complete as to the condition of the ethmoid air-cells and the antrum of Highmore.

If any pathologic process be found at any of these sites, it must, of course, be brought under control.

The appendix is also a possible site of focal sepsis, and should be always eliminated as a possibility in this connection. *X*-ray of the intestinal tract following ingestion of bismuth or barium may reveal anatomic conditions which need attention.

Röntgen-ray examinations have revealed, in a high proportion of cases of arthritis which are not frankly infectious, and even in some cases which are, that the colon may be elon-

gated, rather tortuous, and the seat of stasis. The bismuth test-meal may be notably delayed in its passage through the bowel. These factors are of importance chiefly, perhaps, in the non-infectious type of case where their correction may have beneficial results, but they should have consideration in all obstinate cases. Sometimes the use of proper abdominal support in the form of a belt or corset, when the abdominal walls are flabby and protuberant, may prove of use. Considerable attention has thus been given to the restoration of the proper anatomic relations in such cases, but in general these measures are likely to fall short of the desired mechanical correction, and are useful rather in connection with the other fundamental therapy indicated than on their own account alone.

The contentions of Sir Arbuthnot Lane[84] in regard to the almost exclusive origin of the various arthritides in anatomic and physiologic aberrations of the large bowel have raised much discussion, and much division of opinion. In general, it may be said that the existence of the relations he points out cannot be denied in some instances. However this may be, the treatment he advocates, namely, ileocolostomy and colectomy, is of such severity as to render its general application very limited. There are few surgeons who care to proceed to such extremes, however serious the condition of the patient in respect to his arthritis. Some workers, however, have substantiated Lane's contentions, and reports are not lacking in which great benefit has followed these radical surgical procedures.[85]

Returning to more practicable forms of therapy, it is important that the function of the large bowel in these conditions of stasis be assisted with the mildest medicinal measures which will prove effective, such as, for example, petrolatum, cascara, and so on. The well-known action of agar-agar in acting as a non-absorbable foreign substance to stimulate peristalsis obviously suggests it as a useful help. It may be administered as such, broken up and sprinkled through oatmeal or bread and puddings, or, preferably, in the form of little wafers now to be had at leading grocery stores.* Agar-

* Mansfield Wafers: Made by the Mansfield Laboratories, Inc., 5 Appleton Street, Boston, Mass.

agar absorbs water in the intestines, and so greatly increases in size as materially to increase in volume the intestinal contents.

Another field of attack should be the genito-urinary tract, and careful examination may reveal a boggy prostate, the site of a low-grade infection. Massage of this gland and an examination of its secretion under a microscope may reveal the presence of cellular elements in undue amounts, or even the causative organism at fault. Similarly, the seminal vesicles may harbor low-grade infection. In women the cervix uteri may be the seat of a semipurulent discharge, and the glands of Bartholin may also show infection.

It is, of course, chiefly from the genito-urinary tract that arthritides caused by the gonococcus may originate. Gonococcal types differ not at all in principle from those caused by other organisms, though clinically they differ in attacking principally the larger joints, such as the knee or ankle. Only rarely are the smaller joints of the fingers concerned in chronic cases, unless a secondary infection by streptococci has occurred. Vigorous treatment of the causative gonorrheal infection is, of course, imperative.

The above-mentioned sites are those in which it is most likely that infection may be harbored, but it must be borne in mind that it may exist anywhere in the body, and consideration must be given to all other portals of entrance, such as the bladder, pelvis of the kidney, and so on.

Treatment based upon the above considerations will often give striking results, and is to be strongly urged as the logical procedure to be followed. If the patient's general condition is unsatisfactory for any necessary operative procedure he should be prepared with as little delay as possible to withstand it, since delay invariably means greater damage to the already diseased joints, and very possibly implication of other parts as well. When such steps as the above are being taken, and, indeed, at all times during convalescence, effort should be unsparing to improve the general condition of the patient by means of good food, systemic tonics, fresh air, and such exercise as he is easily capable of without undue fatigue. The gastrointestinal tract may, or may not, be affected in these conditions, and forced feeding can be followed in ema-

ciated cases only within the limits of its easy function. This must be kept actively in mind, since derangement of the stomach through overeating is more harmful than failure to institute forced feeding.

It is common knowledge that in recent years the use of vaccines in a large variety of diseases has been widely exploited, but, perhaps, in few other conditions have they been more used than in the various arthritides. Out of the extravagant hopes and claims for these measures which once obtained, a dispassionate viewpoint has finally been evolved. It seems clear that vaccines have a real, though limited function, in combating the infectious types of arthritis. Fundamentally, as elsewhere indicated, the causative infection must be removed, in order to reach the cause. However, where a joint long remains the seat of the morbid process and the exciting agent has been apparently removed, the intelligent use of autogenous vaccines may be productive of distinct benefit. Vaccines should never be used as a substitute for the more fundamental therapy of removing the cause, but where this is difficult or impossible, or where for other reasons it is desirable to help in the modification of infectious factors, vaccines may serve to increase the resistance of the individual toward the organism at fault.

The use of stock vaccines is less to be recommended, but even with them good results have been reported by some observers. Pending the preparation of autogenous vaccines from a determined focus of infection the use of a stock preparation of the type of organism isolated is sometimes justifiable. Complement-fixation tests may be useful in determining the organism responsible. The use of so-called polyvalent stock vaccines, however, made from a combination of strains of organisms most frequently isolated from infectious types of arthritis at large, and having no necessary specificity to the case at hand, is obviously in the nature of a shot in the dark, and the profession has everywhere properly discredited this.

In administering vaccines certain principles must be kept in mind. The basis on which they act is that in these low-grade infections the economy has been unable to develop a degree of resistance sufficient entirely to overcome the invad-

ing organism. By suitably timed injections of dead cultures of the organism at fault, it may be possible to stimulate the system to a heightened defense, or offense, against it, which results finally in overcoming the development of the organism. From this it obviously follows that only when the mechanism of defense is still active and capable of further stimulation should thought of the use of vaccines be entertained, and it is probably for this reason that their use in the acute overwhelming infections has been disappointing. In pneumonia and typhoid fever the mechanism is probably already stimulated to its utmost. Furthermore, the dosage should be such that a real stimulation is induced. If too little vaccine be used, the effect may be insufficient. If too much be administered, there may be added a further burden to that under which nature is already struggling. The best criterion of the proper dosage is probably the reaction induced, which should be definite, and yet not too great for the condition of the patient. This reaction is characterized generally by some fever, chilliness, malaise, or even an exacerbation of all symptoms, together with soreness at the site of injection, but these evidences should subside within a period varying from some hours to a day or two, depending upon their severity. It must be remembered that, although the type of organism at stake often can be determined, the virulence of the particular strain cannot, without preliminary experimentation; and that prudence demands the use of small doses until the degree of reaction is clearly ascertained. Furthermore, the condition of the patient must be such that there is reasonable belief that stimulation of the body defenses is probable. Just as in the presence of an overwhelming infection the defense of the body is already at its maximum, so in asthenic states the defensive processes may be so low that attempts to stimulate them result only in adding a further burden.

For these reasons no single dosage can be recommended in these conditions, but, in any event, the initial dose should be small (say, 10 million killed micro-organisms), and should be increased with successive injections in proportion to the reaction developed. The interval between injections should be several days—not more than ten—and never less than that required for ample recovery from the preceding dose. Usually the

reactions eventually grow less, despite the increase in dosage, and when the effects of increasing injections have been properly studied, as much as 250 or 500 millions may be employed with benefit. While some time may be required to ascertain the end-result of such measures, they should not be persisted in after reasonable evidence has accumulated that benefit is not to be derived.

Recently the treatment of arthritis by the intravenous injection of foreign proteins has been introduced with some reported success[86] in acute articular rheumatism, so-called, and the subacute variety of arthritis. Gonorrheal arthritis of from two months' to three years' duration has apparently also been benefited. The nature of the reaction effecting these changes is not yet clear to the authors of the method, and its full possibilities and limitations await further investigation. The method practised depends upon the intravenous injection of 2 mils of a 4 per cent. solution of proteose, although in later experiments typhoid vaccine was substituted for the proteose solution.

The vaccine was prepared from the Rawlings strain, killed by heating to 55° C. (131° F.) and preserved by 0.5 per cent. phenol. The dosage varied from 75,000,000 to 150,000,000, and was followed by more or less reaction in the shape of leucocytosis, chill and fever. Injections were given daily in some cases, and at times twelve to twenty-four hours sufficed for the joints to appear normal. No ill effects were reported as a consequence of the injections.

In general, medication plays but a small rôle in these arthritides, beyond the obvious availability of agents such as iron, strychnin and digitalis when indicated. Certain drugs, however, have a larger function, and it would be improper to omit reference to the very real value of arsenic, either in the form of Fowler's solution, 1 to 2 minims (0.07 to 0.14 mils) after meals, or as sodium cacodylate, ⅕ grain (0.013 Gm.) after meals, in a certain proportion of cases. As a general tonic in those rendered anemic by the chronicity of this disease it is of great use, but, more specifically, in incipient cases it may sometimes be sufficient, in conjunction with other steps toward regulating hygiene, to dissipate all symptoms. There are many individuals living upon the borderland of muscular

and joint disturbances of a rheumatoid nature, in whom the interrupted use of arsenic maintains their health. In the writer's experience small doses are best, as subjects of arthritis are sometimes very susceptible to its influence, and show especially early the signs of its therapeutic limit.

The use of the salicylates and aspirin is so well known as to require little emphasis here. Although in the nature of specifics in acute inflammatory rheumatism proper, they are by no means so sure a panacea in the chronic rheumatoid affections, but their influence upon pain is very real in a large majority of instances, and can be depended upon to give the patient comfort while a more fundamental therapy is attempted. During the later stages in hopeless cases they should be held in reserve as instruments of real but limited value, and potential factors of serious digestive disturbance, if improperly used. Furthermore, they not infrequently lose their effect more or less as time goes on, necessitating eventually the increased dosage, which finally upsets digestion. Aspirin* in 5- or 10- grain (0.325 or 0.650 Gm.) doses can be used very happily in advanced cases for those emergencies when the patient desires to make a particular effort, or when an unbroken night's sleep is important. He should be encouraged, however, in all instances to get along with as little as possible.

The iodids have long been in vogue in connection with arthritis, and while it cannot be definitely stated that they are of no avail, the benefit to be derived from them is certainly very small. Their greatest effect presumably follows full dosage, and yet it is of the first importance to avoid any disturbance of digestion to which this therapy may lead.

Thyroid extract is a drug which has accomplished at least temporary benefit in a certain number of cases, but in general its use is ill-advised. It has the property of "hastening metabolism," and in some degree this acts analogously to exercise and the *x*-ray, and may be followed by subjective and objective improvement. These benefits are slight and infrequent, however, and are negatived by the inevitable and dangerous

* Aspirin is the proprietary name for acetyl-salicylic acid, which is cheaper, and of course equally efficient.

nervousness and tachycardia which eventually supervene if its use be long continued.

The use of an extract of the thymus gland has also been advocated.[87] This seems to be entirely harmless, but while some success with it has been reported, later observations have not seemed to bear this out, at least in the writer's experience. The dosage is from 10 to 20 grains (0.65 to 1.3 Gm.) three times a day over a long period of time. It is conceivable that it might have an action comparable to that of thyroid extract.

For many years it has been the custom of the profession to give colchicum and lithia in these joint disturbances which at some period of their progress were formerly mistaken, and, indeed, are now not infrequently mistaken, for gout. Neither is of avail, however, and need not be considered. The use of lithia is based upon erroneous conceptions.

Another method of therapy which has had great vogue, and to which nearly all chronic patients have at one time or another been subjected, is that based upon elimination. It is the practice of some clinicians to induce free action of the bowels, but there is no virtue in the purgation which is sometimes advised.

Whatever the type of arthritis and whatever the portal of entrance to the toxin, elimination can rarely keep pace with the access of fresh toxin, and should never be pushed to the point of debilitating the patient. Water should be freely taken, but only upon the general grounds which make its use always advisable.

One means of inducing heightened elimination has been through the bath and sweat processes, so much and so long in vogue in various sanatoria, both here and abroad. In certain early and borderland cases it is highly probable that the induction of free diaphoresis is of some real benefit, and when coupled with the proper conditions of hygiene and the like it may stem the tide, and cause the subsidence of all symptoms for a long time, if not permanently. More frequently, however, patients who undergo these measures improve at the time, only to relapse when the measures are desisted from and the usual manner of life is resumed. Not infrequently also patients suffer harm, which may be perma-

nent, from too prolonged treatment of this nature. Above all, depleting and exhausting courses of baths and hydrotherapy are to be avoided. They are likely to add to the weakness already present, and to produce a general debility from which the patient may be long in recovering. However, these measures have a real, though limited place, in the therapy of arthritis, provided they are not allowed to replace more important measures directed toward removing the underlying cause as described above.

The joints may also be the subject of local therapy in the form of baking, massage or medicinal applications. These three manœuvres have in themselves no curative value, but they play, nevertheless, a very useful part in conjunction with other more important measures. In most cases baking gives considerable subjective relief, but it is to be practised with due care that the patient is not thrown into a profuse perspiration or weakened by being too long subjected to it. When the joints are the subject of such passive or active motion as may justifiably be attempted during convalescence, baking will be found to render them much more flexible for these exercises, so that more can be accomplished than by manual methods alone.

Massage and passive or active motion have a very real function in these conditions, but they should be controlled by the general principle that further irritation of any already inflamed joint should be avoided. The chief exception to this is to be seen when it becomes necessary in the course of a chronic but quiescent arthropathy to attempt gradual extension of motion in a stiffened joint. When the associated tendons have been the seat of a fibrositis, and through disuse have become shortened, it is obviously necessary that they should be stretched before the muscles opposing them can regain function. In accomplishing this a certain degree of irritation and pain is unavoidable, but this should rarely be induced until the arthritis proper has subsided. One other exception to the above-mentioned principle is to be seen in those cases which are progressive from bad to worse despite all efforts to the contrary. In them a moderate degree of irritation is justifiable, if it retains existing function. Every effort must be made under these circumstances to postpone

the immobilization of joints which will eventually supervene, despite treatment.

Massage itself does not necessarily involve any movement of the joints, and yet, unless care can be exercised, they may be incidentally disturbed, and so be made to suffer by its use. Its most important function is found in those instances where it becomes necessary to restore atrophied muscles to functionating capacity. Sooner or later in nearly all chronic cases of arthritis there is more or less sparing of the joint, and hence of the muscles which move the joint. As a result of this, atrophy of disuse follows in some degree, and, indeed, may go on to nearly complete loss of function. It is perhaps not often appreciated by those in charge of such cases that part of the difficulty experienced by patients in moving a chronically painful joint arises from the fact that great overaction of the reduced muscle substance becomes necessary. This results in irregular and explosive effort to move the joint, which in itself introduces painful factors. Furthermore, the muscle often shares in the systemic processes causing the arthritis, and may itself be the subject of myositis.

The relative violence of such muscular effort is the greater in wasted muscles, so that it is important that the muscle volume and tone be restored as nearly as possible. Massage can accomplish a great deal in this connection if begun very gradually and continued cautiously, increasing only as the patient is able to stand it. Furthermore, if the muscles be not too painful to tolerate it, massage, in a sense, may take the place of exercise, and contribute toward improving the local muscular, as well as the systemic, metabolism. This effect upon the systemic metabolism is very real, and should be utilized in every case where the circumstances of the patient warrant it, and it is not too painful a process for him to undergo with comfort. It is to be borne in mind, however, that, together with exercise, it makes a demand upon the body as a whole, and the physician in charge should see that the food intake of the patient, together with the period of rest allowed for recuperation after massage, is quite adequate to the individual case.

The use of rest in the treatment of the arthritides needs some emphasis. There are some instances in which the con-

tinued normal function of a part may perpetuate locally the symptoms of an arthritis which has elsewhere subsided. Particularly is this true of the weight-bearing parts, such as the foot, knees, hips and sacroiliac joints. In subsiding arthritides of the hand, for example, knitting may serve to introduce mechanical irritation of joints already the seat of an inflammatory process. In nearly all cases, whatever the distribution of the arthritis, rest will achieve some benefit, if it follows upon a period of more or less activity, and, within limits, it can nearly always be profitably advised. On the other hand, it should rarely be carried to the point of adding to the atrophy of disuse, which is so prone to follow upon a chronic arthritis, and, furthermore, if too long persisted in, unnecessary limitation of motion, or even partial ankylosis, may ensue.

When the sacroiliac joints are the seat of an arthritis, particular care is necessary to differentiate the possible and the actual operative factors. Only when these have been clearly analyzed can treatment be intelligently directed, and there is probably more confusion of diagnosis here between the infectious or other arthritides and the mechanical types due to subluxation than in any other joints.

As a purely practical measure attention must be drawn to the importance of flat-foot as a contributory factor in cases of arthritis involving joints below the waist. Discussion of the true static arthropathies has no place here, but, in conjunction with the varieties of arthritis mentioned, flat-foot and the improper distribution of the body weight play a rôle. The feet should be critically examined in all cases, and it is noteworthy that the true infectious arthritides of the feet can be very closely simulated. This influence must be eliminated as a factor or corrected. Prolonged rest, strapping of the feet, casts to hold the feet in proper position, exercises, correct support of the arch, advanced and raised heels, and shoes made on a proper last, may all be useful in meeting the necessities. Their institution should preferably be supervised by an orthopedist, though the possible factor which flat-foot plays here sometimes escapes even the specialist.

Local medicinal application to the joints is confined to a very few measures which have demonstrated value. Hot, cold, or counterirritant applications of any kind may give tem-

porary subjective relief, and are, of course, entirely harmless. The well-known counterirritants of the pharmacopœia can, of course, be applied, but the most useful measures in this connection are perhaps methyl-salicylate ointment and mesotan ointment, the latter in strength of 10 to 25 per cent. Care should be exercised that no undue irritation of the skin be produced by overapplication of these drugs. Wrapping the joint in gauze soaked with a saturated solution of magnesium sulphate may relieve pain considerably.

Climate needs little mention beyond the fact that subjects of arthritis do better in general in a dry and warm climate than in cold and damp localities. Abrupt changes in weather conditions often cause added discomfort to these sufferers.

PRIMARY PROGRESSIVE POLYARTHRITIS.

Finally we come to the last important group of arthritides which deserve consideration here, this being the so-called primary progressive polyarthritis, the fourth group in the classification adopted. Opinions differ as to whether this is really a distinct entity in itself, or whether it is properly a variety of the preceding secondary chronic infectious type of arthritis. However this may be the etiology is by no means so clear as in the case of the infectious type, and consequently treatment has been proportionately difficult and less successful.

The pathology of this group does not differ radically from that last described. The disease may arise insidiously with fever and periarticular swelling of the smaller joints, or it may be characterized rather by an increasing stiffness and pain with less implication of the soft tissues. In both instances it becomes eventually diffuse, and more or less symmetric. The first of these two varieties is sometimes called the exudative type, the latter the so-called dry form.

There is no way of ascertaining clearly when a case is first seen that it belongs in this general group, because the possibility of an infectious origin cannot be dismissed until it is proved not to exist by means of the careful measures outlined above. When every step has been taken, however, and every possible source of infection has been removed, and the patient

still fails to respond in the manner desired, it may justly be suspected that the case is one of this refractory type. Again, there is a certain proportion of cases in which no infection can be found which also, obviously, belongs in the same category. This class calls for the most patient and thorough care on the part of the physician, and it is precisely in this type of case that the various adjuvants of baking, mineral waters, baths, and the host of other measures recommended have been most freely advised.

In these cases are found most frequently the elongation and tortuosity of the large bowel, together with stasis and great delay in the passage of the bismuth test-meal, mentioned under treatment of the infectious arthritides. Even more importantly than in the last-mentioned group should this condition call for correction, and the measures there outlined should be given careful consideration. The radical operations advised by Lane and his followers have here the excuse which comes nearest to justifying them as an established procedure.

Radium emanation has recently come into the foreground as a therapeutic agent in this disease, but it is as yet too soon accurately to outline its limitations for good and evil. It is clear, however, that, as indicated in connection with measures such as arsenic, *x*-ray and thyroid extract, it importantly influences metabolism at large. Its effect in this connection is comparable to that of the *x*-ray. Among the various actions attributed to radium emanation are those of "energizing the body ferments" or enzymes, and increasing tissue oxidation. Speculation is unprofitable here, but it seems clear that clinical results which follow its use can best be explained upon the above hypothesis. Its therapeutic use, however, is essentially limited to those cases where the effects of heightened metabolism within the tissue-cells are sufficient to meet the constant access of toxic matter to the cells; at least, this seems to be the case. The use of the commercial preparations of radium emanation, for which somewhat extravagant claims have been put forward, has met with little real success. In skilled hands treatment by this powerful agent has some value in these conditions, but in general benefits from this source are best sought at the various "Kurorts" of Europe and elsewhere, where they are heightened by the *régime* of life instituted at

such places. In the report of the work carried out at the Radium Institute, London, 1914[88] it is stated that the daily administration of 250 c. cn. of radium emanation solution of a strength of not less than 1 millicurie per liter is sometimes productive of very remarkable results. The degree of improvement is hard to predict with certainty, but the cases which appear to derive most benefit are those in which the disease is of relatively short duration, and the changes are periarticular in type and polyarticular in distribution. At least six weeks are likely to elapse after the institution of treatment before any change is noted. The whole topic of radium therapy is so little developed that dogmatism at this stage is to be discountenanced, and unless the subject be approached through the legitimate and scientific channels of those working in it, it were probably best left alone.

For some years the writer has made the general group of arthritides now under consideration the subject of a study, which has resulted in measures capable, at times, of very real benefit. These studies are not yet complete, and conclusions based on them are therefore subject to later modification, but in view of the definite benefits observed it seems proper to include here such references to the method as will at least allow an understanding of the principle concerned, and the general manner in which it should be applied. For full details the reader must be referred to the original articles on the subject.[89] It is not entirely clear that this general type alone has yielded to the treatment to be described, and, on the other hand, there is some reason to regard the second subvariety of it, called the dry form of primary progressive polyarthritis, as possibly more refractory, but for convenience of discussion the question at large can be taken up here, since for this general type of arthritis no other specific therapy has been advanced. Indeed, when every possible focus of infection has been removed in any thoroughly studied case, and the patient still remains the subject of an active arthritis, these measures can be properly considered. In explanation of this method of treatment a few preliminary remarks are necessary. It has been observed, in individuals the subject of rheumatoid arthritis who have undergone a major operation, that following

the operation there is often a period of subjective relief. It has long been known that agents that profoundly influence the body metabolism, such as arsenic, thyroid extract, and the *x*-ray, sometimes have a temporary beneficial effect upon the objective as well as the subjective symptoms of the disease. It has seemed, from a correlation of these facts, that this benefit following operation might be due to institution of the so-called basal metabolism, approximated in the period of starvation which follows many abdominal operations. It was conceivable that under these conditions the body might be spared the utilization of the food intake, and that, at the same time, having less to do, the tissue-cells most concerned in these processes would be able better to perform their work. This hypothesis was in harmony with the beneficial effects in this disease of arsenic, the *x*-ray, radium, exercise, and other agents which operate in the opposite connection; namely, to hasten catabolic processes, and, possibly, anabolic processes as well, and, so to speak, "whip the flagging horse."

If these facts were true, it seemed possible that there might be a midpoint at which the subject of arthritis could utilize a lowered intake of food with satisfaction to his nutritive needs, and, at the same time, without injury to the joints, as expressed by the arthritis. The general correctness of this hypothesis has been indicated by work based upon it.

Apparently there exists, in many cases of this general type of arthritis, a level of intake of carbohydrate and protein food above which the individual is ill, and below which improvement or entire convalescence may follow. This level varies with the individual case, and no set rule can be given for ascertaining it, though it is generally below the average intake of either; often very much so.

Experiments in connection with fat indicate that, although it is rarely possible to replace the curtailed caloric intake entirely by fat, it is often possible to give part of this in the form of fat without injury to the arthritis, and with benefit to the nutritive needs.*

* Since the above was written it has been found possible to carry through to convalescence two chronic cases who were made to gain weight by the use of large quantities of fat which entirely replaced the caloric deficit.

In brief, the facts seem to be that in treating cases along these lines it is necessary to reduce the carbohydrates by an important amount, varying with each case; that it is necessary to reduce the proteins considerably, but relatively not so much; and, finally that, although the fats cannot be handled with impunity, they may be used with caution to increase the caloric value of the ingested food, and meet, in some degree or altogether, the loss of weight.

It is entirely conceivable that the rôle played by the foodstuffs depends upon bacterial activity in the intestinal tract. It seems likely at present that at least some of the substances causing the damage arise in the cleavage of the carbohydrate and protein molecule, either bacterially within the intestines or physiologically from enzyme action, and that an important phase of the disease concerns the ability or inability of the tissue-cells, particularly perhaps in the muscles, to utilize and to destroy these midproducts. Whatever the real method of action, the fact is that reduction of the carbohydrate and protein reduces the sum-total of injurious matter which reaches the tissues.

In undertaking the treatment of a case it is the writer's practice to observe the individual for a few days, during which a record is kept of the entire food-intake in terms of grams and mils. This can readily be done by any nurse trained in modern methods, and requires nothing more than a measure and a pair of scales weighing to 1 gram. Some intelligent patients have conducted this alone. From this record a pretty close idea can be obtained as to what the individual normally ingests, and about the number of calories required to keep him in equilibrium. During this probationary period he is urged to live nearly as possible according to his usual habits, with relation to exercise and general activity.

It is well to have the ingredients of the probationary diet prepared according to some definite formulary. A very useful one is to be found in a small book entitled "Food Values,"[90] in which are set forth nearly all the useful table articles, with their caloric value appended.

From the record of the individual's intake of food, the average daily caloric intake can be fairly closely approximated. Since the symptoms of disease have persisted in gen-

eral under these conditions, it follows that any method of treatment which involves a curtailment of food must reduce the food-intake to a point at least below the average which has been ascertained.

Furthermore, it is of prime importance that the nutritive needs of the body be met or kept well in mind, and in making reductions as severe as those sometimes entailed here it is important that no harm be done to the individual. One can be sure of this only if the actual caloric value of the intake ordered is known. It is important to give as much food as the subject can tolerate without injury to his joint-structures, from which it follows, in the writer's experience, that diets have had to undergo several changes, the first, or even the second, being too ample to achieve the results desired; but as it is impossible to determine beforehand what that amount will be, it is well to proceed cautiously in the above manner, especially as some few cases of long standing and apparently severe arthritis manifest striking improvement upon relatively slight modifications in their diet.

It may be said that a recent case in a young subject requires in general a slight modification, as compared with an advanced case of long standing, and if the average normal food-intake be found high, it may be assumed with fair probability that the new diet need not be very restricted. On the other hand, if the average food-intake be found low, it is obviously necessary to strike an even lower level. For example, in patients living upon a caloric intake of 3500 calories there is a very good chance that a reduction of this amount to, say, 2500 calories, will still leave them ample for all reasonable nutritive needs, and at the same time relieve them of a surplus of food amounting to 1000 calories. On the other hand, if the individual be ingesting normally only 1900 calories, as sometimes occurs in the secondary invalidism of this disease, it is plain that no such reduction can be made. It is necessary then, however, to strike an even lower level of, say, 1500 or even 1200 calories, and so on. There is obviously a limit to which this can be carried, except for short periods. The use of calories in this connection is, of course, simply as a measure of amounts of food, and has no necessary relation to the study of calorimetry of the body in general. In draw-

ing up a new dietary the protein and carbohydrates are the chief factors to be considered, and the caloric value decided upon must begin with the calories from these sources. Certain other factors are also important, however. For example, there are many articles of diet whose bulk is considerable for the food-value contained, such as apples, tomatoes, spinach, celery, beets, cabbage, turnips and lettuce, which are exceedingly useful to take the edge off the appetite and to serve as a basis for the really nutritive, but restricted articles which follow in the form of bread, eggs and meat.

It is immaterial how the carbohydrates and proteins be administered within the given amounts determined upon, though a nearly exact estimation is easier if they be simply prepared. Until convalescence is well underway, it is best to adhere to the simple staples, such as bread, butter, sugar, milk, roast, broiled or boiled meat and fish, rice, boiled or roasted potatoes, etc. No one diet can be chosen that is suitable for all cases, for the reasons given above, but as an illustration the following may be cited as a dietary suitable to a mild type of case. It must be emphasized, that no one diet will suit every case:

		Calories.
Breakfast:		
1 apple	150 Gms.	72
1 egg	50 Gms.	83
1 slice bread	30 Gms.	81
Butter	10 Gms.	80
Weak coffee and 20 per cent. cream, 1 tablespoonful	15 mils	54
Sugar 1 teaspoonful	7 Gms.	28
11 A.M.:		
Olive oil, 1 tablespoonful	15 mils	121
Luncheon:		
Vegetable soup, strained, f℥vj	180 mils	25
Lettuce	q. s.	
Mayonnaise, 1 tablespoonful	15 mils	187
Spinach	50 Gms.	28
Butter	5 Gms.	40
1 orange	250 Gms.	96
4 P.M.:		
Olive oil, 1 tablespoonful	15 mils	121

Supper:		Calories.
1 apple	150 Gms.	72
Weak tea and 20 per cent. cream, 1 tablespoonful	15 mils	54
Sugar, 1 teaspoonful	7 Gms.	28
Chicken or beef	50 Gms.	100
Bread, 1 slice	30 Gms.	81
Butter	10 Gms.	80
Lettuce	q. s.	
French dressing, 1 tablespoonful	15 mils	148
9 P.M.:		
Olive oil, 1 tablespoonful	15 mils	121
		1700

The beneficial influence of a diet cannot always be pushed to a successful conclusion for a number of reasons: for example, in the presence of emaciation, anemia, advanced heart disease, and the like, the already unstable equilibrium may be further upset by a lowered food-intake, however slight, and this may then be strongly contraindicated. It is, of course, of the highest importance that individuals should be put at modified or complete rest coincidently with a moderate or severe curtailment of their energy intake, and this important essential cannot be emphasized too strongly. The weight also must be watched daily.

Patients often need encouragement during the long period required for treatment, since they not unnaturally mistake the secondary results of arthritis for a continuation of the causative arthritis, although, in fact, this may be subsiding.

It must not be supposed that all cases will respond equally well. Wider experience may develop more types in which no response can be detected, but it is safe to say that in a large number, perhaps in a majority, of cases which have yielded to no other measures, definite benefit or cure can be seen. The general nutrition of the individual largely determines the degree to which treatment can be pushed, and in every case the individual, and not the arthritis, must primarily determine the vigor of treatment. It is of small benefit to improve the arthritis at the expense of the patient's health as a whole. The writer cannot emphasize too strongly this point, for any severe dietary procedure is a two-edged tool, and must be used with the greatest possible caution.

In undertaking treatment along these lines it is important to determine whether arrest of the disease in an advanced case would justify such prolonged sacrifice as must be made to achieve a real cure. It not infrequently happens that structural deformity is so far advanced and muscular atrophy has become so chronic that, although the arthritis were to be arrested instanter, there would still remain practically as much disability and pain as originally noted. It is as true of treatment here as in any other condition, that there must be an intelligent selection of those cases which best deserve an effort. In relatively young persons of good nutrition, in whom no foci of infection exist, especially if they be not very far advanced, the most vigorous attempt is more than justified, and it is precisely in this class that the most significant results can be seen.

The writer has found codliver oil of great value after response to treatment has been clearly evidenced, and the patient has settled down to protracted adherence to a low diet. Under these circumstances, when the toxemia has been removed, the blood may rise from figures of anemia to those of complete normality.

As convalescence proceeds, it is often possible to increase the food-intake, in some cases very measurably, so that the necessity for reduced activity may grow less or disappear, and the individual may be allowed to resume his full activities.

Rheumatoid Arthritis or Arthritis Deformans. The condition designated by these names is classified by modern nomenclature under Arthritis, especially of the infectious type and therefore calls for no separate consideration, as the above terms are merely generic synonyms.

Myalgia. Myalgia may be defined as a condition in which there is pain in the muscles. Outside of such causes as traumatism and the like it may be regarded as referable to the conditions which form the underlying basis of arthritis in general. In other words, in arthritis of the infectious and "metabolic" types the nerves and muscles may be affected as well as the joint structures *per se,* constituting the condition commonly referred to as myalgia.

The condition within the muscles is regarded by Llewellyn Jones (see Arthritis, p. 544) as of the nature of a fibrositis.

The muscle may show nothing abnormal to inspection or palpation except tenderness. On the other hand, a sense of resistance and rigidity may be detected. Pressure, passive motion, and active motion may be exceedingly painful. Any of the muscles of the body may be attacked, although perhaps those most frequently affected are in the lumbar region, the upper arm, and the shoulder.

The *treatment* of this condition depends upon recognition of the underlying cause and the institution of steps to remove it. The cause may be purely infectious, as in a decayed tooth or an inflamed tonsil. If every possible infectious focus is removed from consideration the cause may be conceivably regarded as "metabolic," in the sense described under Primary Progressive Polyarthritis. Under these circumstances treatment can be instituted along the lines mentioned under this topic (see p. 559).

Local therapy has a place in myalgia, and in cases of trifling severity is often sufficient to dissipate it. Massage may be agreeable, if it does not cause too much pain, but it should be governed by the same principles which govern massage of the joints. Heating, baking, and the well-known counter-irritants are entirely admissible, and the salicylates by mouth are often of great avail.

In general the subject of myalgia should be regarded as forming one of the manifestations of the causes producing arthritis, and so treated.

CHRONIC VILLOUS ARTHRITIS.

The villous arthritis of Goldthwait probably is a purely static type often referable to flat-foot, for example, and needs no particular emphasis here other than that sufficient to direct attention to removing this possible cause. It is characterized by an absence of general symptoms, by crepitation, and by varying degrees of pain upon movement.

CHRONIC ARTHROPATHIES OF THE SPINE.

The spinal types of arthropathy do not differ in principle from the infectious and primary progressive varieties, and are characterized by the imprint of the disease upon the spine

rather than upon other structures. The vertebral column is subject to the same morbid process which affects the other joints, and for purposes of treatment it seems unimportant to differentiate the pathologic variations encountered.

It is sufficient that attention be given most critically to the discovery of possible etiologic factors in the way of infection, and when this fails of results the treatment must depend upon the general principles already described. Under these circumstances the influence of diet as outlined under the fourth group of primary chronic progressive polyarthritis may be very real. It is possible, however, since the natural range of motion of the vertebral articulations is slight, that conditions approaching ankylosis may supervene sooner in the spine than elsewhere, and treatment should be instituted proportionately early.

STILL'S DISEASE.

The general principles which underlie the consideration and treatment of the above arthritides apply to Still's disease, so called; and in this condition every effort should be made to ascertain the etiologic factors at stake. In certain features, such as the glandular and splenic enlargements, it differs from the diffuse arthritides of adults, but the pictures of the adult and juvenile forms of arthritis may closely resemble each other. Except for the limitations which childhood places upon all therapeutic measures there is but little reason to particularize in regard to treatment of this condition. Restrictions of diet in general are not advisable, owing to the necessities for growth. The paramount importance to the child of good hygiene and good food must be kept prominently in mind. Mutch has laid emphasis[91] upon the rôle of the *Staphylococcus citreus* within the intestine and in the circulating blood as the cause of Still's disease, and advocates the radical procedures of ileocolostomy and colectomy as the only measures of value.

HEBERDEN'S NODES.

Attention has long been directed to the articular deformities known as Heberden's nodes, but it is very questionable whether they deserve any particularization. Indeed they

seem to be merely local expressions of the overgrowth seen in a variety of arthritides that affect the smaller joints of the hand. Their presence means the existence at some time of the underlying factors which produce infectious or primary arthritis.

Not infrequently a bursa may be the seat of pain and swelling, and under these conditions it seems clear that its synovial lining suffers from the diffuse process attacking other structures. Subsidence of this swelling takes place as the cause of the general morbid process is removed.

SUBCUTANEOUS FIBROID NODULES.

Subcutaneous fibrous nodules are not infrequently met with in the course of an arthritis, and may respond by subsidence to the institution of measures which benefit the arthritis proper.

This has been the cause to a noteworthy degree in some instances treated by the writer along the dietary lines discussed under primary chronic progressive polyarthritis.

Frequently neuritis is an accompanying feature of arthritis, and, indeed, alone may be an expression of the underlying factors which in other cases lead to disturbances in the joints. It tends to improve as the cause is removed. It is probable that a not inconsiderable number of cases of sciatica have a similar pathologic basis.

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Diseases of the Nervous System

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Diseases of the Nervous System.

FOREWORD.

In the ensuing Section on the Treatment of Nervous Diseases, the authors have endeavored to lay stress upon simple physiologic procedures. These are still too little appreciated by the profession, and too much reliance is still placed upon the giving of medicines. The importance of rest, exercise, massage, full feeding, careful attention to the digestive tract and the various organs of elimination are insisted upon. The importance of psychotherapeutics is pointed out, and the various methods discussed as occasion requires. Finally, whenever medication is of value, this also is considered in detail.

PART I.

FUNCTIONAL DISEASES.

Before undertaking the treatment of a given disease, we should have before us, whenever possible, clear clinical conceptions. This is especially true in the field of the functional nervous diseases, and we will, therefore, first briefly review the symptoms of the various affections before entering into the details of the treatment. Further, inasmuch as neurasthenia is expressive of a generalized type of functional nervous disorder, it will be given a rather full consideration. Such a course leads to clear and direct indications for treatment. The latter, as we will see, apply also to a number of other, and in a sense allied, affections. In the discussion of these, the general principles indicated in the study of neurasthenia will frequently be referred to.

Among the various generalized neuroses that present themselves no less than five conditions should be clinically differentiated. They are as follows:

Neurasthenia,
Neurasthenoid states, or psychasthenia,
Neurasthenia symptomatica,
Hysteria, and
Hypochondria.

NEURASTHENIA.

This affection is commonly spoken of as nervous exhaustion, or nervous prostration. The symptoms are found to depend on chronic fatigue; and Dercum applied to it, years ago, the term, the fatigue neurosis. Notwithstanding, it continues to be much misunderstood by many physicians. Thus, some claim that nervous exhaustion is rare as a primary affection, that it is always secondary to some other disease, such as tuberculosis; that it is the result of some condition requiring surgical interference, or that it occurs only in those predisposed from birth because of a defective physical and nervous organization. A brief consideration will soon reveal the erroneous character of these views. To begin with, neurasthenia may present itself as a primary state of persistent exhaustion, and may occur in an individual otherwise normal; or it may occur in individuals in whom there is a more or less marked hereditary element, *i.e.*, a neuropathic constitution or make-up, and in which the symptoms consequently assume a special character. The condition resulting is spoken of as a neurasthenoid state, or, to employ the term devised by Janet, as a psychasthenia. Simple or uncomplicated neurasthenia, *i.e.*, nervous exhaustion, occurring in previously healthy persons, offers an exceedingly favorable prognosis. The neurasthenoid states, or psychasthenias, on the other hand, from the very fact of an underlying defective nervous system, are much less promising, although in many cases marked improvement follows the application of general physiologic methods.

Prominent among the causes of neurasthenia we find overwork, insufficient rest and sleep, mental anxiety and worry, and improper nourishment combined with the excessive use of tea and coffee, and perhaps alcohol. We are not all born physically or mentally equal; we cannot all be athletes physi-

cally or giants intellectually. The attempt to compel the organism to do more work than it was ever intended to do eventually breaks it down or exhausts it. A very common cause, especially in America, is the constant strain and habitual excess of work imposed by the high pressure of business. Students not infrequently break down, especially at the close of their examinations. Much, too, has been said of sexual excess. Probably the drinking, carousing and loss of sleep which so often accompany sexual excess, play here an important rôle. Excessive and frequent child-bearing with prolonged lactation, combined with overwork or worry, is also a potent cause. Prolonged nervous exhaustion likewise at times follows infectious diseases, especially influenza.

The essential *symptoms* of neurasthenia are those of chronic fatigue, and are classified as sensory, motor, psychic and somatic.

Sensory Symptoms. Among sensory symptoms the patient presents a general fatigue sensation, or weariness, which is very persistent. It is one of the primary symptoms, and may dominate the picture as long as the disease exists. Likewise the patient may complain of being dizzy without any real signs of vertigo. The dizziness is probably due to a vasomotor weakness, so pronounced that slight changes of position affect the intracranial circulation.

Localized sensations of fatigue, such as headache, backache, or limbache are frequent. In early cases they disappear on rest; in long-standing cases they may only be relieved if the rest be long-continued. The headache is diffuse, and is usually most pronounced in the occipital region, though it may be frontal. With the headache we frequently have associated sensations of pressure or constriction of the head, drawing sensations in the back of the head and neck; or heaviness and fullness may be complained of.

The backache is usually referred to the lumbar region, sometimes to the midscapular region, and at times to the sacrum or coccyx. Like the headache, the backache may be regarded as a fatigue sensation. Sometimes the back becomes sensitive to touch, or there may be tenderness over the spine, limited to various areas. The physicians of a previous generation termed this symptom "spinal irritation." Occasionally

there are areas of tenderness of the scalp, gums, or teeth, but never the localized tender spots met with in hysteria. The aching of a particular part of the body often bears a distinct relation to the occupation of the patient, and the overuse of a special group of muscles; for instance, aching in the legs of persons who stand or walk a great deal, or aching in the arms in persons with occupations in which the arms are used excessively. Loss of sensation—anesthesia—it is important to state, is never present, though the patient may complain of numbness. Paresthesias, such as tingling or sensations of heat and cold, may also be complained of.

Symptoms relating to the eyes are frequently present. These may consist of weakness and fatigue of the eye muscles or irritability of the retina. Quite commonly they are accompanied by headache. Because of the underlying exhaustion, refraction of the eyes usually fails to relieve either the ready fatigue or the headache.

Again, the patient may state that he cannot hear well, and that he frequently fails to understand what is said to him. The fact, however, is that because of his exhaustion he is unable to concentrate his attention. True deafness does not exist. On the other hand, auditory hyperesthesia is rather a common symptom, and is expressive of the general nervous irritability of the patient. Fatigue symptoms affecting smell and taste are also usually present, but are usually less pronounced; however, some neurasthenics complain excessively of various odors, or of the taste of various articles of food.

Motor Symptoms. The primary motor symptom is, again, ready exhaustion. There is, however, no local weakness, and nothing resembling paralysis. Tremor is often present, and is a fine intention tremor. There may also be noted at times slight muscular twitchings, in small muscular bundles here and there; for instance, in a few fibers of the frontalis of one side, or in one or both orbiculares palpebrarum; or in other facial muscles.

The tendon reactions are usually slightly exaggerated, though they may be normal. Now and then the response may even be diminished. Ankle clonus has been noted, but it is, as a rule, faint and rapidly disappearing. A Babinski sign is never present.

Psychic Symptoms. The most striking psychic symptom is a diminished power for continued mental effort. Brain-work sooner or later brings on symptoms of exhaustion, and the patient finds it increasingly difficult to concentrate his attention. This symptom may greatly alarm the patient, giving him the idea that he is about to lose his mind. The patient is likewise unable to think readily. There is a lack of spontaneity of thought. This the patient himself realizes, for he declares repeatedly, "I cannot think." Further, the tired man has not the same will-power or aggressiveness; he soon begins to hesitate, often to be uncertain, and to lack decision. Again, there is a markedly increased irritability. That the tired man is usually a cross man is a familiar fact. This irritability is expressive of a diminished self-control, a diminished inhibition. Finally, the patient who is chronically tired is also afraid; weakness and fear go hand-in-hand. He may suffer from sudden attacks of fear. These attacks are spontaneous, apparently causeless in origin, and generalized in character. They may be accompanied by pallor of the face and palpitation of the heart, just as is normal fear. At times the physical signs are very pronounced; thus there may be sudden weakness, pallor, coldness of the surface, excessive tachycardia, and even relaxation of the sphincters. Such attacks may be mistaken for hysteria, but they bear no resemblance to the latter affection.

Attacks of fear occurring in a patient previously neuropathic may give rise to special forms of fear, such as the fear of being alone (monophobia), or the fear of crowds (anthropophobia); or they may give rise to various obsessions and morbid anxieties. The states resulting have been described under the head of neurasthenic or neurasthenic-neuropathic insanities, while Janet has devised for them the convenient term, psychasthenia.

In addition to the other psychic phenomena, the patient frequently suffers from insomnia. Some neurasthenics cannot sleep for a long time after retiring; others fall asleep readily and promptly, but awaken at intervals through the night. Quite commonly the patient falls asleep from exhaustion toward morning, but this sleep is unrefreshing, and on awakening the patient feels tired and depressed. Quite commonly,

too, the disturbed sleep of neurasthenia is accompanied by repeated micturition.

Somatic Symptoms. The somatic symptoms, like all of the symptoms of neurasthenia, are those of ready exhaustion. Thus, the gastro-intestinal symptoms are those due to atony and deficient innervation of the stomach and intestines. Digestion is delayed and enfeebled, and constipation is the rule. The circulatory symptoms consist in a lessening of the force and modifications of the rhythm of the heart's action, of the character and frequency of the pulse, and of changes in the vasomotor tone. Palpitation of the heart is a common symptom; at times it is associated with the digestive disturbances, at others, with attacks of fear. Coldness, dampness, and lividity of the hands and feet are also common symptoms.

Disturbances of the secretions are also present, likewise dependent upon deficient innervation. Thus, the skin is quite commonly moist, especially of the hands and feet, which may be cold and clammy. At times, again, the patient sweats excessively upon relatively slight exertion. At other times the skin is unusually dry. The urine, too, may reveal changes, such as an increase or a decrease in volume. Changes are also noted in the character both of the sweat and of the urine, but these are secondary, and cannot detain us.

The sexual phenomena of neurasthenia are, again, those of weakness and irritability. Diminished desire and power, premature ejaculation, diminution of the sensations normally present, are among the common symptoms complained of. In addition, others may be noted, dependent upon the sex and the habits of the patient; for instance, in young unmarried men, excessive frequency of seminal emissions, and in young women the occurrence of the orgasm spontaneously during sleep.

TREATMENT.

The treatment of neurasthenia resolves itself into three factors: the application of rest, the administration of food, and the elimination of waste substances. The last indication is met by attention to the action of the skin, kidneys and bowels. The methods used will necessarily have to be adapted to each

patient individually. If the exhaustion is the result of overwork or overstrain of the nervous system, this must, of course, be corrected, and excesses of all kinds must be guarded against. These measures of themselves afford in many instances decided relief. However, in the majority of cases, the institution of rest as a therapeutic measure is imperative. Rest has a very wide application, and may vary very greatly in degree.

Partial Rest Methods. If the neurasthenia is not too severe, or if the patient finds himself in such circumstances, financial or otherwise, that he cannot take a complete rest, we must use so-called partial rest treatment. As a matter of fact, we are forced to adopt this method in a very large number of cases.

The patient should be instructed to diminish his work as much as possible, at least so much as is unphysiological, and at the same time increase his hours of rest to the fullest extent possible. He should retire not later than 9 o'clock, and rest in bed as many hours as his work will permit, *e.g.*, he should, if possible, spend ten hours in bed. After his midday meal he should lie down again for one or two hours. Both morning and afternoon, however, he should, if possible, exercise gently in the open air, but never to the point of fatigue.

Along with attention to the hours of rest we should take up the question of diet. The food should be such as will be easily digested, and in as large amounts as possible. Milk especially should be added to the diet. Many neurasthenics claim they cannot digest milk, and in these cases the patients should begin by taking very small amounts after meals, between meals, and at bedtime. Taken in this manner the quantity can be gradually increased until the patient is taking six full glasses of milk daily in addition to his other food. Red meats are stimulating, but, as a rule, do not agree with neurasthenics. The excessive use of starches and sweets is likewise to be avoided. The white meats, fish, chicken, oysters and eggs should be taken freely, as also the succulent vegetables. Spinach, lettuce, celery, ripe and stewed fruits are valuable. Whole cereals, as wheat or oatmeal, and bran-breads are also indicated. In other words, a liberal mixed diet, with the addition of milk and eggs, is the best for a neurasthenic. All stimulants, alcohol, tea, coffee and tobacco, are contraindicated. Under certain conditions, it may be necessary to allow

a cup of coffee or tea in the morning, but usually a cup of cocoa or hot milk will be found to be equally acceptable to the patient.

Many of the vague aches and pains which attack the neurasthenic are doubtless due to faulty elimination and the retention of waste substances. In order to increase the elimination of these materials, we should increase the amount of liquids the patient consumes. Most neurasthenic patients drink too little water, and it is well to order a fixed and rather large amount to be taken at regular intervals between meals. It may be added, in this connection, that milk is not only one of the most useful foods for these patients, but being a liquid food it is also a valuable aid in elimination.

The skin should be stimulated by bathing; a brief immersion bath, a shower or a sponge bath, followed by a gentle rubbing, will answer the purpose. A warm bath in the evening, just before retiring, is both relaxing and restful to the patient, and is often helpful in relieving the insomnia from which so many patients suffer. Because of the general weakness and inability to react to cold plunge baths, they are not indicated in neurasthenia. However, cold sponge baths may be used in the mornings just after rising. The application of the cool sponge and rub-down stimulates the patient at the time his energy is at its lowest ebb. Elaborate apparatus is not necessary to apply hydrotherapeutics in these cases.

Massage may be employed, and in a measure takes the place of exercise. It is likewise a valuable aid in combating insomnia, and, of course for this purpose it is given just before the patient retires at night.

Electricity sometimes proves to be of benefit in some cases. Quite commonly it acts merely by suggestion, but occasionally it appears to have a distinctly stimulating effect. Static electricity is the form most commonly employed.

Full Rest Methods. The neurasthenia may be so pronounced that it becomes necessary, if at all feasible, to put the patient to bed upon absolute rest. To obtain good results close attention must be paid to the details of the treatment. The rest must be as complete as possible, and the treatment is of course best carried out away from home, under absolute isolation. The patient is to lie quietly in bed, sitting up

merely to take food, and leaving the bed only to empty the bowels or bladder. A number of weeks of such rest is usually sufficient for ordinary cases, but at times a much longer period is required. When the exhaustion is profound, the patient must be fed by the nurse, and should even be turned in bed in the endeavor to save the patient all exertion. Mental excitement must be avoided, and relatives and friends should not be permitted to visit the patient. Further, all correspondence should be eliminated. In other words, complete isolation of the patient, with only the physician and nurse in attendance, is desirable. Necessarily this rule must be modified at times, according to the case. However, most patients make better progress when the isolation is maintained.

In employing full rest methods, massage is indispensable. In the beginning the massage should be used gently, and for a short time only. Severe and deep massage may increase the fatigue. Gradually, as the patient improves, the massage may be increased to one hour in duration. Passive exercise also proves very helpful.

Electricity is not as valuable as massage. It is useful to stimulate the muscles, and is indicated toward the end of the rest period, that is, preparatory to getting the patient out of bed. The slowly interrupted faradic current is the one usually indicated. The current should be so applied as to evoke in the various muscle groups a certain number of contractions. This treatment should not continue over twenty to forty minutes.

The diet in neurasthenia has already been outlined under partial rest methods. It is wise to begin with a moderate amount of food. At times it may be best to begin with milk alone, 4 to 6 ounces (120 to 180 mils) at meal-times, between meals, and just before the hour for sleep. In most patients, however, some solid food can be given in the beginning of the treatment.

White meats should be preferred. Vegetables such as spinach, lettuce, celery, squash and later peas and string beans may be added until a full diet is reached. Potatoes should not be given for some time, and then not in large quantities. Wheat bread also should only be permitted in limited amount. The neurasthenic needs a mixed diet, one that will supply all

that the tissues require, but the full diet must be approached gradually.

The milk should be slowly increased in quantity, until the patient takes from 8 to 12 ounces (240 to 360 mils) six times daily. Often the patient objects to milk, either because he dislikes it, or because of inability to digest it. In such cases the milk may be modified in various ways. A little salt added to the milk will in some instances be helpful; in others the addition of some alkaline waters, such as Vichy, Apollinaris or plain soda-water. Further, the milk may be predigested, or some digestive powder added to the milk just before it is taken. Buttermilk is valuable, especially when the patient suffers from constipation. Whey also may be used, though its nourishing qualities are of course greatly inferior to that of milk. Kuymiss, *i.e.*, imitation kumyss, is of much more value than whey, and is frequently well digested when milk, even modified, fails. At times it becomes necessary to discontinue milk altogether, and then egg-feeding may be resorted to. Eggs are best given raw. The egg is broken into a glass, leaving the yolk whole. The patient can soon learn to swallow it with a single effort. If necessary, a pinch of salt or a few drops of lemon juice will render the egg more palatable. The patient should begin with 1 egg between meals, this being gradually increased so that the patient after a time takes 6 or more eggs daily.

The neurasthenic, at rest in bed, can digest and assimilate large amounts of food. Great care must be given to the digestive tract; especially must constipation be guarded against. The skin also should be kept active by sponge bathing, and massage should be given thoroughly. As a rule, a rapid increase of weight is noted under these circumstances.

The patient should have a special nurse, who devotes all of her time to the patient. In the case of male patients, it is of course necessary that the nurse should be of the same sex. The nurse should sleep in the same room, or one adjoining that of the patient. The patient should be prepared for a period of absolute quiet between the hours of two and four in the afternoon; many patients learn to sleep during this time. It will be found most convenient also to have the nurse take her hours off for recreation and change at this time. It is

needless to add that the nurse must have both patience and tact, and must in her conversation direct the patient's thoughts away from her ills and into pleasant and healthful channels. It is best also that the nurse herself should give the massage. A strange masseuse coming in for this purpose often disturbs the patient, and in this way may retard her progress.

If the details of the treatment are properly carried out, the patient will gradually improve. There is a decided gain in weight, the circulation improves, the extremities cease to be cold, the muscles become firm, and the color of the skin gradually assumes the appearance of health; the nervousness and restlessness begin to disappear, and little by little the patient feels at ease, comfortable and relaxed. Sooner or later, however, if the treatment is successful, some return of spontaneity is observed. The patient becomes more active mentally, and the inclination to exert himself becomes manifest. As soon as the maximum amount of improvement has been reached, as is indicated by a cessation in the increase of weight, and by a rather pronounced desire on the part of the patient for activity, we should begin getting the patient out of bed. This period is not reached before six, eight, ten or twelve weeks have passed. The patient should at first sit up for from five to ten minutes once or twice daily. Very gradually this time should be increased until five to six hours out of the twenty-four are spent out of bed. Light passive exercise, Swedish movements with resistance, and, later, calisthenics, should be instituted.

Walking in the open air or an occasional drive should at this time be permitted. The time out of bed being steadily increased, the patient is out of bed the greater part of the day; rising at 10.30 in the morning, lying down between two and four, and going to bed soon after the evening meal. A little later the time is still further increased, but the patient is instructed to still take his breakfast in bed, and to lie down between two and four. Finally it becomes necessary to send the patient away. Preferably he should not go directly to his home, but should go for a period of about two weeks to the seashore, country, or mountains. During this period the patient may gradually resume an ordinary, every-day plan of living, and begin resuming communication with friends. It is

important to emphasize the fact that after a course of rest treatment it is very important that the patient should adopt some regular form of exercise, especially out of doors. One important point, however, must be borne in mind, and that is, that the exercise should always stop short of fatigue. Such a course leads to permanent and durable results. A strictly physiologic mode of life should, of course, be insisted upon. If possible, the patient should be induced to take up some agreeable and useful occupation. Work within physiological limits is beneficial to both the mental and physical condition of a patient. Indeed it may be questioned whether a high level of health can be maintained without it.

The reader will observe that thus far nothing has been said of medication, and indeed in many cases no medication will be required. At times the insomnia may be so severe as not to yield to massage or hydrotherapy, *e.g.,* the warm bath or drip-sheet. In such instances it may be necessary to use medicines, such as the bromids, luminal, medinal, trional, sulphonal or veronal. These remedies should not, however, be continued long, nor should they be given in large doses. The best results are often obtained by not giving the same drug continuously, but by changing from one to another. Morphin is rarely, if ever, necessary and chloral also is rarely indicated. The milder remedies above enumerated as a rule answer every purpose.

Little also has been said of psychotherapy, and it may be here remarked that only such psychotherapy as is contained in the suggestion that the patient is getting well is of value. Neurasthenia has as its basis an underlying physical condition, and, until this is corrected, suggestion is of but little avail. To be sure, cheerful and bright surroundings are of value here as in other affections, but no reason exists for the employment of hypnosis, psychanalysis, or other special psychotherapeutic procedure.

At times it may be necessary to prescribe tonics, such as arsenic, or iron, especially if surface pallor be marked, or at times strychnia may be indicated.

HYSTERIA.

Hysteria may be defined as an innate neuropathy, the various symptoms of which present the intrinsic evidence of a mental origin, and which is characterized by a pathological susceptibility to suggestion, and by an emotional instability.

Heredity is a strongly predisposing factor. In many cases there is a family history of hysteria; sometimes of other nervous affections. Charcot maintained that hysteria was always hereditary. It may occur in either sex and at almost any age. Persons of an emotional, impassionable and non-resisting make-up more frequently manifest its symptoms than others. That the patient is innately neuropathic is revealed by a brief study of the symptoms.

In early times the symptoms of hysteria were ascribed to disorders of the womb. The name of the affection is indeed derived from ὑστέρα (hystera), the Greek word for womb. The Greeks believed that in an hysterical attack the womb becomes detached from its moorings and goes wandering about the body seeking sexual satisfaction. It was not until the seventeenth century that it was described by a French physician (Charles Lepois) as a nervous affection, and it was not until the latter part of the nineteenth century that its symptoms were carefully studied by Charcot, Paul Richer, and Gilles de la Tourette. It was Babinski, however, who established the true nature of hysteria. Babinski failed in one hundred consecutive cases of hysteria, not *previously examined* by physicians, and in which he carefully avoided suggestion, to note in a single instance the presence of hemianesthesia. Evidently this symptom, which up to his day had been one of those most frequently observed in the clinics, was produced in the patient by the suggestion of the physician's examination; that is, it was an artefact. This proved to be true of all the other symptoms of hysteria. In other words, in hysteria the various symptoms observed are the result—of course, inadvertent—of the suggestions presented by the medical examination, or they are the result of suggestion derived from other sources.

Again, it is found to be impossible to elicit a given symptom such as anesthesia in a normal individual, even when

direct suggestion is employed. Evidently the hysterical individual is pathologically vulnerable to suggestion, *i.e.*, he is the sufferer from a pre-existing and innate neuropathy.

In Charcot's time the symptoms of hysteria were elaborately classified and charted, but since the introduction of Babinski's method—that is, of examinations without suggestion—these symptoms have to a large extent disappeared from our clinics. Such symptoms as are still met with have their origin in other sources of suggestion than medical examinations, or in medical examinations that have been improperly or unskilfully made.

Bearing in mind the purely mental character of the *symptoms* and their origin in suggestion, it is easy to understand that the symptoms elicited bear no relation to the facts of anatomy; the symptom of anesthesia in a given case, for instance, presents no relation to the facts of nerve supply and distribution. Equally the phenomena met with frequently stand in crass contradiction to the well-known and established facts of physiology.

It has been the custom, especially in past years, to divide the symptoms into sensory, motor, psychic and visceral phenomena. These we will briefly enumerate, bearing in mind, however, that they are observed, if at all, typically in much-studied and long-standing cases.

Sensory Symptoms.—Anesthesia, hypesthesia, paresthesia, or hyperesthesia may variously be noted. The areas involved present no relation to nerve distribution or to spinal segmentation. The anesthesia may involve a hand, like a glove, and in such instance is spoken of as a glove-like anesthesia; or a foot and leg, like a stocking, *i.e.*, a stocking-like anesthesia. Sometimes a mere segment of a limb is anesthetic, or isolated patches may be distributed over various parts of the body, *e.g.*, the face, trunk, as well as the limbs; or the anesthesia may affect one-half of the body, being sharply limited by the middle line. Usually all forms of sensation are involved, but in some cases a dissociated loss of sensation is present, *i.e.*, a loss of the pain sense or the temperature sense without loss of the tactile sense. There may be isolated patches of hyperesthesia or hyperalgesia, located over the spine, beneath the breasts, such as areas of inframammary tenderness, or over the groins,

namely, areas of inguinal tenderness; or it may be over any other part of the body, head or limbs. The tenderness is not genuine, is always superficial, and disappears upon deep pressure. The areas are found more frequently on the left side of the body. At times they are found on the mucous membranes, even in the vagina and rectum.

As regards the special senses, *e.g.*, the eye, contraction of the visual field may be elicited. In keeping with suggestion, contraction of the field is developed on the same side of the body in which there is also a hemianesthesia. In former years elaborate studies were made of "contracture of the color fields," but these, like contracture of the visual fields in general, are now known to be artefacts.

Similarly hysteric deafness may be elicited. This is usually incomplete, the hearing being merely impaired. Bone conduction is well preserved. There is generally associated anesthesia of the external auditory meatus, often of the drum, and at times of the auricle. Loss of smell and taste may likewise be elicited in hysteria.

Motor Symptoms. The motor phenomena of hysteria express themselves as palsies, contractures, tremors and incoördination.

The palsy may take any form, hemiplegia, paraplegia, or monoplegia. With the palsy there is frequently associated anesthesia of the paralyzed part. Contractures in hysteria rarely simulate the contractures seen in organic disease, though at times the palsy may simulate a paraplegia or hemiplegia. The tremor usually consists of to-and-fro oscillations varying from four to twelve per second; however, more frequently the rate is from seven to nine in a second. The tremor does not resemble either that of paralysis agitans or of multiple sclerosis.

Incoördination may express itself as astasia abasia; *i.e.*, the movements of the patient will betray no ataxia when lying in bed or while sitting, but when the patient attempts to stand or to walk incoördination makes its appearance, and usually becomes very marked. The gait does not resemble either that of tabes or of cerebellar disease. It is extremely irregular; wide, oscillatory, coarse or grossly bizarre movements of the legs, arms and trunk are commonly observed. The tendon

reflexes in hysteria do not present constant phenomena. They may be somewhat exaggerated, though never, unless the patient has been trained by suggestion, to the degree seen in organic disease. They are seldom diminished, and never really lost, though here a caution is necessary, as some persons in apparent health never have a knee-jerk. The latter is also normally absent in many children. Similar remarks apply to the Achilles-jerk and to the ankle clonus; a persistent ankle clonus may, however, though rarely, be noted. The skin reflexes may not be at all modified, though they may be diminished. They may even be lost in an anesthetic limb. A Babinski sign is never present.

Somatic Symptoms. Among the visceral manifestations of hysteria we note vomiting, rapid pulse, vasomotor disturbances, rapid breathing, cough, yawning, retention of urine, anuria, phantom tumor, aphonia, spurious aphasia, and other bizarre phenomena.

Hysteric vomiting is often associated with anorexia nervosa, *i.e.*, with nervous loss of appetite. Pain may be complained of, and may lead to the erroneous diagnosis of organic disease. When circulatory disturbances are present, such as tachycardia, flushings, pallor, coldness, lividity or dermatographia, their functional nature is at once apparent. Occasionally hysteric rapid breathing may be present. Frequently this symptom is unassociated with any change in the pulse rate; there is no cyanosis, dyspnea, or cardiac distress. Hysteric cough is accompanied by no physical signs; the cough may assume a bizarre form, and may suggest the crowing of a cock or the yelping of a dog. Hysteric yawning is usually frequently repeated, exaggerated and prolonged.

The sphincters are not involved in hysteria. The patient may claim, among other things, that she has anuria, but the grave symptoms of suppression of urine are never present. Polyuria is frequent; in such case the patient passes large quantities of pale urine of a low specific gravity.

The abdomen may at times be greatly distended, or the appearance of pregnancy simulated. The distention may be irregular or limited in outline, and give rise to a so-called "phantom tumor." Needless to say, examination soon reveals the nature of the symptom; the ballooning proves to be due to

gas-distention and the tumors to localized spasms of the abdominal muscles.

Psychic Symptoms. The psychic state in hysteria has already been sufficiently indicated; suffice it to repeat that, owing to his innate neuropathy, the patient reacts abnormally, and at the same time suffers from a marked emotional instability. Paralysis, convulsions, nausea, vomiting, blindness, all have their origin in suggestion. Hysteria, too, may be contagious; symptoms may be communicated from one hysterical patient to another. The mental attitude is always introspective, and the patient manifests an inordinate craving for sympathy.

Sometimes hysterical crises supervene. These present symptoms which vary greatly. The latter may consist of light emotional storms attended by laughing and crying, or by transient alterations of conduct, the emotional character of which is apparent. On the other hand, the attack may be more pronounced. In such case, it is usually preceded by a period of depression and irritability, during which the patient for slight causes weeps or laughs. This stage may last for several days, and hysterical symptoms, already present, become more marked, or new ones make their appearance. Sooner or later a convulsion ensues. There is first a tonic spasm, including the muscles of the trunk and extremities. Unlike epilepsy, this tonic spasm may be quite prolonged. Sooner or later, however, it is followed by a clonic spasm, which after a time subsides. The patient is never unconscious. At times the patient passes through a series of bizarre motions, contorting the body into various positions, suggestive of volition or purpose, or the patient may assume dramatic or passionate attitudes, accompanied by cries and weeping. Later the patient becomes quiet and may fall asleep.

At times the rigidity becomes so pronounced as to constitute a catalepsy. At others the patient passes into a condition of ecstasy. At other times still the patient may pass into a condition of lethargy, or the somnambulism, or the sleep of hypnosis may be simulated.

TREATMENT.

The physician in his examination and treatment of a case of hysteria should be careful that by suggestion he does not induce new symptoms or make worse those already present. Elaborate and prolonged examinations, frequently repeated, and especially if made by different physicians, are baneful in their effects. A careful physical examination should of course be made of every medical case, but care should be taken that no inadvertent, indirect or other suggestion be conveyed to the patient that might be harmful in its effects. On the contrary, the examination should be conducted in such a way as to suggest to the patient that there is nothing very serious the matter. On the other hand, the physician should frankly admit the existence of the symptoms from which the patient complains, at the same time giving the patient the impression that he does not regard the symptoms as of great consequence. It is important when marked stigmata, such as anesthesia or paralysis are present, to lay as little stress on them as possible. The patient is usually encouraged by being told that the symptoms are not dangerous, and that she will make a good recovery. However, hysteric patients are frequently very jealous of their symptoms, dwell upon them insistently, and are anxious to impress the physician with their severity. Needless to say, all the patient's complaints should be listened to patiently. It is only in this way that confidence is established, and it is only by establishing confidence that the physician will be able later to influence the patient by suggestion. Tact, patience, and a judicious sympathy must be maintained throughout. Finally, the examination should be made largely from the standpoint of internal medicine. The patient does not believe herself to be hysterical, and to tell her so often does great harm. It should never be forgotten also that hysteria is now and then present, when there is also actual organic disease; especially may we meet with this complication in girls and young women.

If in a given case the hysteria be severe, the patient should, if possible, be put to bed, and a course of radical rest treatment instituted. This will give the physician an opportunity to relieve the symptoms, the result of the patient's unphysiologic habits of living, and the sense of comfort and well-being that

results from a properly conducted rest-cure is of itself a powerful suggestion of a returning health. Isolation is here of the greatest importance. Otherwise, the comments and solicitous sympathy of well-meaning but mistaken friends and relatives make nugatory the best efforts of the physicians.

It is of course very important that the nurse should be in full possession of the details and nature of the case, that she should be endlessly tactful, gentle and firm, and that above all she should at all times keep up the suggestion that the patient will get well. No hard-and-fast rules can be given to govern the conduct of the nurse, save to carefully observe the instructions of the physician in every detail, and to keep him informed as to the changing aspects of the case.

The details of the rest treatment have been fully considered in the treatment of neurasthenia, to which section the reader is referred. However, symptoms at times arise requiring special attention, and often necessitating a modification of the general plan of treatment. It may be that the patient centers her thoughts on one special feature of her case, such as a painful area, a palsy, vomiting, or retention of urine. Here success often depends upon the resourcefulness of the physician and the conscientious co-operation of the nurse.

Painful sensory areas should be treated by massage, or by hot and cold douching. At times, various forms of electricity are helpful. A nurse skilful in giving massage may "rub out" the painful area, and, occasionally, a placebo, such as a capsule containing starch, when given coupled with the suggestion that it will give relief, is effectual. Inguinal tenderness may be very persistent, and give rise to the belief in the existence of some ovarian trouble or appendicitis. However, the measures here indicated, if persisted in, will usually prove successful.

In the case of a palsy, the patient should be encouraged to make the effort to move the affected part, and also taught to do so. Suggestion proves in such instances very serviceable; *e.g.*, the patient is told she is succeeding, and that the muscles are becoming stronger each day. In such cases, too, massage and electricity are of decided benefit, and are a valuable aid to the suggestion. If the palsy be a monoplegia, it is well to have the patient make some desired movement with the unparalyzed

member first, and then to try the same movements with both limbs. Many times a paralysis rapidly disappears after a skilfully made suggestion.

It need not be stated that the chief benefit of massage and electricity in a hysteric palsy is due to the suggestion. The faradic current may be all that is necessary. However, the static spark or the high frequency applied by means of a vacuum electrode are doubly suggestive, as their application is *visible* and impressive. Palsies as well as contractures are at times exceedingly difficult to combat. In these cases, passive movements, in addition to electricity and suggestion, are also of benefit. In the experience of the writers, contractures are more difficult to treat than palsies. If contractures persist for too long a time, a certain degree of actual fixation may take place in the joints and fibrous tissues. Under such circumstances it is proper to overcome the condition by free movements, and, if necessary, giving at the time an anesthetic. Contractures, with few exceptions, disappear while the patient is asleep, or while he is under the influence of the anesthetic. However, at times actual changes have taken place in the joints and fibrous tissues, and in these cases the contracture persists. As soon as the condition present is fully ascertained, treatment by passive motion should be given daily, combined with suggestion, and the control of the will over the affected limb should be stimulated as much as possible.

Astasia abasia, or hysterical ataxia, should be treated by exercise of precision, *i.e.*, by efforts at retraining, together with the judicious use of suggestion.

Vomiting, nausea, and loss of appetite are difficult symptoms to treat. In anorexia nervosa the articles of food which the patient objects to or refuses to take are generally those which she needs the most. At times everything is objected to, and the patient takes and retains only very small quantities of nourishment, if any. Usually she declares that the taking of food produces nausea; even the taking of liquids may be followed by eructations, belching of large amounts of gas, and retching and marked distention of the abdomen. At times, indirect suggestion is very helpful. Thus the milk or some other article of food may be emphatically and ostentatiously forbidden. Again, the nurse having been previously in-

structed, may mention a given article, and in reply the physician should treat it as of no consequence. In such case the patient, inasmuch as the article of food is not being forced upon her, may ask the physician whether it could not be tried in her case. This is more likely to be the result if the amount of food taken has been grossly insufficient. Notwithstanding, indirect suggestion frequently fails, and in such instances both the physician and the nurse must rely upon their tact and persuasion. It is unnecessary to add that in making an agreement with a patient, the latter must be rigidly adhered to. Occasionally the suggestion that the patient will retain the food if she swallows it in large quantities is successful. Of course this also may fail, and the physician may be forced to give the patient exceedingly small quantities, and in such physical condition that it cannot be vomited; *e.g.*, thoroughly hard-boiled, partially dried and powdered yolk of egg, bread crumbs, crackers, and the like. Exceedingly small fragments of bacon may be added, or salt judiciously added. Water may be freely administered by high enema or by the Murphy drip.

Other articles of food may then be tried, especially milk. Exceedingly small quantities only should be attempted, and repeated at frequent intervals. If milk is rejected, white of eggs or albumin water may be tried. Again, the milk may be modified in any manner to suit the individual case, according to the judgment of the physician. If a beginning can be made the patient will soon retain other food, such as finely minced ham, dried beef, minced chicken, steak, and the like.

If there be marked epigastric tenderness, it may be wise to omit massage of this region for some time, and then very gently and gradually include it.

Usually it is best to avoid medicine, as this also will be vomited. However, it is not infrequently found that bromids are retained. At such times, 20 grains (1.3 Gm.) of ammonium bromid with a little aromatic spirits of ammonia and peppermint water may be given, well diluted. Again, patients sometimes do well under the influence of small doses of morphin, that is $\frac{1}{32}$ of a grain (0.002 Gm.). This may be repeated every half-hour until $\frac{1}{8}$ grain (0.008 Gm.) has been given. Larger doses may be used, but their likelihood to produce and make worse the nausea must be borne in mind. The morphin

may be given dissolved in water, in a few drops of brandy, or in a teaspoonful of iced champagne. Sometimes small doses of cocain may advantageously be substituted for the morphin. Champagne by itself we have not found to be very successful in relieving anorexia nervosa. However, sometimes it will be retained when all foods fail. Carbonated waters may also prove of value.

When no food is retained for several days, it may become necessary to resort to tube-feeding by the nose or mouth. The patient may complain of difficulty of swallowing. It is frequently necessary to resort to this expedient but once. At other times, the mere preparation for the feeding is often sufficient to stimulate the patient to retain nourishment. If tube-feeding fails, it is well to precede the next attempt by a hypodermic injection of morphin, $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008 to 0.016 Gm.) and scopolamin $\frac{1}{200}$ to $\frac{1}{100}$ grain (0.00033 to 0.00065 Gm.). This quiets the patient, and facilitates retention. At other times recourse may be had to rectal suppositories of opium. Rectal feeding also may be resorted to, but usually proves unsatisfactory. Besides, its use may confirm the patient in the belief that she really has some serious affection of the stomach.

It is a remarkable fact that patients suffering from anorexia nervosa frequently preserve a remarkable appearance of health in spite of the persistent rejection of food. In such instance it is probable that food is being taken surreptitiously. In other instances, however, there is a more or less decided loss of weight.

Rapid breathing in hysteric cases is usually of no significance, and as a rule subsides spontaneously. Hysteric retention of urine also is not a serious complication. Rupture of the bladder, of course, never occurs, nor even a dangerous distention. Placing the patient on the vessel, and having within hearing the suggestive sound of running water, the nurse leaving the room for the time being, is often efficacious in inducing the patient to empty the bladder. The catheter is rarely, if ever, to be used. Hysteric anuria is to be treated by the administration of diuretics, and also large quantities of liquids. The anuria is not accompanied by alarming symptoms, and is frequently only simulated. Hysteric polyuria does not require treatment.

Insomnia will usually improve on rest, bathing, full feeding, but if it is necessary to resort to drugs, the bromids, 20 to 30 grains (1.3 to 2.0 Gm.), are the most useful, or luminal, in doses of 1½ to 3 grains (0.1 to 0.2 Gm.), proves very efficacious. At times medinal may be given, in 5- to 10-grain (0.32 to 0.65 Gm.) doses. As a rule, however, a capsule of starch or some other placebo, accompanied with the suggestion that it will induce sleep, answers the purpose admirably. At times it may be wise to use a starch capsule on alternate nights. Very frequently the use of warm baths, just before the patient retires, is productive of good results. At times, too, the drip-sheet or other simple form of hydrotherapy is beneficial.

In many cases of hysteria, a systematic rest treatment cannot be carried out, and in such cases we must rely upon proper physiologic methods of living, together with suggestion. In this connection it is important to remember that occupation, both mental and physical, is of the greatest benefit, though many hysteric patients are disinclined to work, especially those whose means do not necessitate it. Success here largely depends on the resourcefulness of the physicians.

Special psychotherapeutic measures other than those here outlined are not indicated in hysteria. Hypnotism is rarely justified since it does not cure the underlying condition; indeed, it emphasizes the susceptibility to suggestion from which the patient already suffers. Psychanalysis has of late years attracted considerable attention. It has, however, never gained a secure foothold in this country, and, like other fads, is again disappearing. It is a common experience with physicians—with neurologists and practitioners in general—that it is of the utmost benefit to allow the patient to give a full and complete account of his case, to talk himself out concerning his symptoms, their history, causes and allied matters. This fact was recognized long before psychanalysis was thought of. Merely in the course of the patient's story, the symptoms lose in the patient's mind their importance, and often fade away. There is a sense of satisfaction and relief in having told the doctor everything—in unloading everything, as it were, on the doctor. Hysteric patients do not do well unless they have gotten into close touch with the doctor, and have acquired full confidence in the latter. The patient is then in a position to

receive the greatest possible benefit from explanation, advice, and suggestion. However, to inject into the patient's mind, as do the psychanalysts, thoughts of a sexual nature, to give to every symptom, dream, or what-not a sexual explanation, is obviously pernicious and harmful. Indeed much injury is sometimes done by arousing in the patient's mind ideas of self-blame and sinfulness, ideas which often center about sexual transgressions that never occurred, or, in the absence of a positive history, are relegated by the psychanalysts to mythical peccadillos of childhood.

HYSTERIA FOLLOWING ACCIDENTS. "TRAUMATIC" HYSTERIA.

Our views concerning the nervous manifestations which not infrequently make their appearance after accidents have, in the course of years, undergone a radical change. This change has been due to our increasing knowledge of hysteria. The symptoms observed were early embraced under the term "railway spine." Erichsen made use of the term "concussion of the spine." Erichsen, Leyden, Westphal, Erb, and others originally believed that the symptoms were due to a chronic meningomyelitis, multiple foci of disease in the cord and brain, and other organic lesions. No evidence was forthcoming from actual observations to prove the correctness of these views. Owing to the fact that death did not take place—a fact the significance of which was not at the time recognized—cases did not come to autopsy. However, in two instances in which the patients died of intercurrent affections, one of aneurism and the other of acute alcoholism, a careful microscopical study of the brains and cords by Dercum, in 1895, failed to reveal any lesions whatever. Moeli, Wilks, Walton and Putnam pointed out the psychic or hysteric nature of the symptoms. Thomsen and Oppenheim, while admitting the hysteric interpretation of the symptoms, made reservations as to the partial existence of organic lesions, but Charcot showed that the symptoms have no anatomical basis whatever. Charcot pointed out that the symptoms are exactly the same as can be produced by hypnotic suggestion, that they were the result of autosuggestion, and finally that they were all due to hysteria, and nothing but

hysteria. Page also early recognized the non-organic character of the symptoms. It is a significant fact that the terms "spinal concussion" and "railway spine," once in use the world over, have disappeared from the courts and from the reports of physicians for many years past. Oppenheim substituted for them the expression "traumatic neuroses." Unfortunately the word "neurosis," vague and indefinite in meaning, failed to convey any conception as to the nature of the condition present. Notwithstanding, the term "traumatic neuroses" rapidly became the vogue. Soon, however, the hysteric nature of the symptoms became more and more evident, and the expression "traumatic hysteria" came to be used as a substitute. For a long time the profession were inclined to hedge with Oppenheim, as to the purely hysteric character of the symptoms, and such terms as "traumatic neurasthenia," and hybrid expressions as "traumatic hystero-neurasthenia," came to be employed, but they finally gave way with our increasing knowledge to the name "traumatic hysteria," and thus medical men came to adopt a position which one of them, Charcot, had long anticipated.

As we have seen in the preceding pages, the symptoms of hysteria have their origin in suggestion. In the hysteria observed in accident cases the same fact obtains. The suggestion may have its inception in the knowledge of having passed through an accident, re-enforced, it may be, by fright, or, as is most frequently the case, it may have its origin in the possibilities and prospects of compensation. Certain it is that trauma of itself never causes hysteria. It is a noteworthy fact that trauma occurring during sleep, during surgical anesthesia or alcoholic intoxication, is never followed by hysteria. Again, to show the necessary presence of suggestion, we need only cite the well-known fact that persons injured during sports, in gymnastic exercises, in foot-ball, never develop hysteria. Further the hysteria in a given case following an accident bears no relation to the character or the degree of an injury, and the surgeon is not living who can say after examining a bruise, a dislocation, or a broken bone, that the patient will also suffer from hysteria. Finally, it is a notorious fact that quite commonly, even when the hysteria is pronounced, the evidences of injury are exceedingly trivial, such as a small abrasion or a

trifling bruise, or they are actually non-existent and wholly imaginary.

If it is claimed that an injury must in addition be accompanied by fright in order that hysteria may supervene, it must be remembered that fright unaccompanied by any injury whatever is a cause of hysteria. Try as we may, no rôle can be assigned to trauma, and the expression "traumatic hysteria" so frequently and so glibly used by medical witnesses in the courts, describes a condition which does not exist.

How great a rôle the presence of the right to recover damages plays is revealed by the history of railway accidents. Until the recent enactment of workmen's compensation laws, traumatic hysteria was an affection limited to passengers. Physical injuries, of course, were found in locomotive engineers, firemen, brakemen and conductors, but hysteria never. However, when the right to recover damages is present, the immunity of railroad employees disappears. For instance, in Germany, where a system of workmen's pensions exists, railroad employees form a not inconsiderable number of the cases of "traumatic" hysteria, and often prove to be among the most persistent of the pension seekers.

The hysteria observed in litigants bears, as we have seen, no relation to trauma; neither does it bear any relation to fright. Hysteria evoked by fright alone—*i.e.*, hysteria into which compensation does not enter—presents a very different clinical history from the hysteria of litigation. Fright hysteria is of immediate onset; its symptoms supervene at once, at the time the fright is experienced, and, as a rule, it is of short duration, and rapidly subsides. When it does not rapidly subside, or, having subsided, recurs, special causes are at work to bring about its prolongation, and, in litigation cases, it is the prospect of compensation.

In the hysteria of litigation cases, the history is quite commonly that the supposedly injured person is attended by bystanders, conveyed to a nearby drug-store, perhaps sent to a hospital, or taken to his home. In either case he comes under the care of physicians. Soon a lawyer is consulted, medical experts are called in, elaborate examinations are made. How full of suggestion such examinations are, the preceding pages have already shown. Elaborate tests are made, many notes

are taken, strange scientific terms are used; and the connection, in the mind of the patient, that he has been seriously hurt grows steadily in strength, and with this his proportionate expectation in the amount of his damages.

At times it does not occur to the plaintiff until hours, days, weeks, and even months have elapsed after an accident that he has been hurt. In short, in the hysteria presented by plaintiffs, the phenomena observed are due neither to trauma nor to fright, but to litigation, and the proper designation for the condition is "litigation hysteria."

In the interval pending the trial and final settlement of the claim, the symptoms resist every possible form of treatment. This is an absolutely uniform and unvarying experience. It is a noteworthy fact, further, that if an expected trial be not reached, or be for some reason postponed, the symptoms become less marked, and often largely subside until the next date of trial approaches, when they again become more pronounced; fresh examinations are made, the plaintiff becomes worse than ever, and even new symptoms make their appearance. During all of the period pending trial, the patient continues under medical care, but with the settlement all medical attendance ceases; the symptoms disappear, the patient either forgetting all about them or no longer making a voluntary effort to maintain them. If, however, settlement be delayed, the plaintiff neither gets well nor improves, and this situation may continue indefinitely, sometimes for years, as long as any hope of settlement persists in the plaintiff's mind. The plaintiff knows that the success of his claim depends upon the existence and persistence of symptoms. The hysteria of accident claimants thus has its origin in the psychology of compensation, and nothing is of avail save the definite disposal of the claim for or against the plaintiff.

In closing, we must remember that after an accident—*e.g.*, a collision—only a small percentage, perhaps a single individual, subsequently develops hysteria. As a rule, the persons affected reveal in their histories—often carefully concealed, denied or perverted—a previous hysteria, confirmed in character, or they present the crass evidence of the underlying neuropathy already described in the preceding pages.

THE NEURASTHENOID STATES (PSYCHASTHENIA).

The conditions which were at one time classed under the head of neurasthenic insanities have been termed by Janet collectively, "psychasthenia." Dercum has applied to them the term, the "neurasthenoid states." There are here present two factors, one a neuropathy pre-existing, innate and usually hereditary, upon which a second factor, a nervous exhaustion, has been superimposed. Because of this pre-existing neuropathy, the symptoms of the nervous exhaustion present differ from those met with in simple neurasthenia; for example, as a result of nervous exhaustion an individual otherwise normal may suffer from an attack of fear such as has already been described. The attack remains generalized in character, and subsides without any special features being developed. However, if the patient be previously neuropathic, a pathological association may be formed in his mind, so that the emotion of fear becomes definitely linked with certain relations of his environment. Thus, such a patient having a spontaneous attack of fear while he is alone, may subsequently be afraid of being alone, *i.e.*, he acquires a monophobia. All of the symptoms of a psychasthenia are susceptible of kindred explanations. Weakness of will, indecision and lack of inhibition are natural outgrowths of the neuropathy and the exhaustion. A normal neurasthenic, because of his fatigue, loses his usual readiness of decision; in a neuropathic neurasthenic this indecision may become so pronounced as to lead to a true insanity of indecision, a *folie du doute*. In a case of simple neurasthenia, the will-power of the patient may be lessened, but in a neuropathic patient the will may be so weakened that he becomes unable to perform comparatively simple and ordinary acts; thus a clergyman may be unable to mount the steps to his pulpit. Such a symptom is termed abulia. Again, the simple neurasthenic has impaired self-control, becomes irritable, his impulses are not controlled as in health; but this deficiency of self-control in the neuropathic neurasthenic may give rise to special gestures, exclamations, words or phrases which the patient is unable to inhibit; *i.e.*, such a patient may develop psychomotor tics, or *tic convulsif*, as it

has been termed by the French. These movements may consist of bowing or of bizarre gestures, or the patient may suddenly utter disconnected phrases, oaths and obscene expressions. Inhibition having broken down, the symptoms tend to become in time confirmed and established.

The neurasthenoid states, the psychasthenias, do not as a rule offer an encouraging prognosis. However, all means at our command for raising the general health of the patient to the highest possible level should be instituted. It is significant and important also to remember that many psychasthenic persons undergo spontaneous improvement, or have more or less prolonged periods of remission. Others again improve as they grow older, especially is this true as middle age is approached.

In recent cases, full-rest methods with hyperfeeding, massage, bathing, exercise, gradual retraining, and re-education yield most gratifying results. If a tic be present, systematic exercises with difficult or complex movements necessitating careful muscular co-ordination and concentration of will and attention are often of value. However, in long-standing cases little can, as a rule, be achieved.

Psychotherapy may also be used in like manner as in hysteria, though in cases of psychasthenia the results are not as satisfactory. Occasionally if the patient is able to recall the full details of the first occurrence of a special fear or other symptom, the pathological association formed at the time may be broken up; *i.e.*, it may be explained away. If such a result is possible, it can as a rule be achieved in a single interview. It does not, as stated by the psychanalysts, require upward of three years of daily interviews and questionings, nor is it necessary to search for imaginary or forgotten sexual transgressions.

It is an interesting fact that a certain number of psychasthenics present the symptoms of hypothyroidism; especially are they noted in cases presenting marked indecision and abulia. In a few cases actual myxedemoid symptoms are present. In these conditions small doses of thyroid extract, given over a long period of time are of value.

HYPOCHONDRIA.

Hypochondria is a nervous affection, almost equal in importance to neurasthenia or hysteria, although less frequently met with. Occasionally it has been confounded with these affections. Medical writers on the whole have been loath to grant to hypochondria a definite position in our nosology, and for the reason that hypochondriacal states may occur in other affections such as the prodromal periods of melancholia or paranoia, or may complicate other mental diseases. However, hypochondria presents a characteristic clinical picture. Its symptoms owe their origin to a change in the general sense of bodily well-being, a change which gives rise to a more or less fixed conviction of bodily illness. The patient usually seeks for an explanation in the disease of one or more organs. The most careful clinical examination, however, fails to reveal anything of moment. Hereditary factors are very commonly found in the family history. Hypochondria occurs more frequently in men than in women, more frequently in single than in married persons, and more frequently before 40 than afterward.

Hypochondria is a neuropathy which has as its expression a constitutionally diseased personality.

The patient is much concerned in regard to his health. Frequently he is chronically afraid of catching cold, or of some serious affection of the chest. In such case he may wear an excessive amount of clothing. Frequently, too, he is peculiar regarding his food. At one time he eats an excessive amount of meat, at others he is a vegetarian. At one time he diets himself so rigidly that he takes too little nourishment, at others he eats excessively.

He may complain of various vague sensations, such as pains in the head or about the heart, trembling of the stomach or intestines, numb sensations, distress or pain referred to some special organ, such as the liver, or it may be the genitals. At times also he speaks of burning sensations in the mucous membranes, of strange feelings in the skin or hair. Physical examination always yields a negative result. Occasionally there is present a slight atonic indigestion and constipation; perhaps at others coldness or slight lividity of the hands and

feet. The symptoms are, however, always trivial and of little consequence.

The patient notes his condition very carefully, and frequently observes his bowel movements or urine in great detail. Very often he keeps a record of his symptoms, and in the consultations with his physician frequently produces little pieces of paper upon which he has made numerous notes. Quite commonly he gives an account of having visited numerous physicians, remembers the various diagnoses that have been made in his case, declares that none of his physicians have benefited him, and, of course, believes himself to be a very sick man. Occasionally he reads medical books or quack literature, and goes to the physician with the diagnosis of his disease already made. He is almost always taking medicine of some kind, and when he believes he is not obtaining proper treatment from physicians, treats himself. The shelves and closets of his room are usually filled with medicine bottles.

Hypochondria does not always pursue an even course. Ideas of ill-health are at times less pronounced, and actual remissions may occur. In quite a large number of cases the disease fades with advancing years.

Other things equal, the prognosis is most unfavorable, when a neuropathic family history is pronounced. Further, the hypochondria of youth offers, on the whole, a more favorable outlook than the hypochondria which makes its appearance in adult life. The prognosis as to the lucidity of the patient is uniformly good. The patient's mind remains clear; dementia is not established.

In addition to a hypochondria general in its character, we may distinguish two special forms, namely, the gastro-intestinal form and the sexual form. In the first some slight but real digestive disturbance may be present; in the second form, which is more common in young men, there is frequently a belief or fear of impotence, associated perhaps with a history of masturbation or of seminal emissions. Very often these patients are about to marry. As a rule, real impotence does not exist, though every now and then fear and nervousness and the belief in impotence lead to failure.

The indications for treatment are not so clear as in neurasthenia or hysteria. It does little good to tell the patient his

trouble is imaginary, but we should examine the patient thoroughly, point out to him the absence of organic disease, and the purely functional character of the symptoms. Such patients like to be examined, and a properly conducted examination will beget confidence in the physician. However, they are but little influenced by suggestion. The general health should be maintained at as high a level as possible, by attention to hygiene and physiologic living. This being accomplished, the patient should, if possible, be kept at work. The work should be such as to keep his time well filled, and to give him but little time to dwell upon his troubles. It is found that these patients usually enjoy the best health when working the hardest. Hydrotherapy, electricity and massage may be used, but care must be taken not to develop in the mind of the patient the idea that his symptoms are serious.

HEADACHE.

Headache is, of course, a symptom and not a disease. However, it is such an important symptom, and the dominant feature in so many affections, that it requires special consideration.

Headache is either organic or functional. Organic headache is associated with structural disease of the cranial contents, *e.g.*, brain tumor, is continuous, and is associated with sleep disturbances, vomiting and gross physical signs of disease. Syphilitic headaches are, of course, associated with a specific history and other symptoms of syphilis. They are usually more pronounced at night.

Functional headaches may be classified as follows: first, headache associated with the great neuroses, neurasthenia and hysteria; second, headaches of diathetic, toxic and infectious origin; third, headache associated with affections of the special sense organs, such as the eyes and the various viscera; and, fourth, headache associated with various diseases of the blood.

Neurasthenic Headache. Here the pain is, as a rule, dull and diffuse, and suggests fatigue. It may even, when pronounced, quickly disappear upon rest. As a rule, it is not diffused over the entire head, but is located in the occiput and

in the upper part of the neck, or over the frontal region, or just above the eyes. Occasionally the pain is referred to the temples and parietal region, but occipital pain is the most frequent. The pain is often accompanied by a feeling of pressure or constriction (a feeling of a tight band about the head), or is associated with pressure and drawing sensations at the back of the neck. Other sensations, such as heaviness and throbbing, are sometimes described.

The patient presenting neurasthenic headache presents in addition all of the cardinal symptoms of neurasthenia, and the diagnosis of course resolves itself into the recognition of the underlying neurasthenia. Its treatment is the treatment of neurasthenia, and the reader is referred to the section on this subject.

Hysteric Headache. This form of headache is found more frequently in women. The patient complains in terms which suggest exaggeration, while her appearance does not convey the idea of serious suffering. If asked to indicate the seat of the pain, she is apt to point to a small area, often a spot, which can be definitely covered with the tip of the finger. She usually describes the pain as deep or boring, as though a nail were being driven into the head; it is this symptom which has given rise to the term clavus hystericus. On examination this area is found like the other sensory stigmata of hysteria to be sensitive to superficial pressure. Other symptoms, such as ringing or throbbing noises in the ears, and various bizarre sensations, may also be complained of. Hysteric clavus is, of course, of no more significance than the hyperesthesia noted beneath the breasts, over the groins, over the sternum or over the spine. The treatment of hysteric headache is, of course, the treatment of hysteria.

Diathetic Headache. Patients who are afflicted with a gouty or rheumatic diathesis frequently suffer from headache. The presence of the other symptoms of these affections establishes the diagnosis. Diathetic headaches occur more frequently in persons of middle life. Hereditary factors, too, are sometimes present.

Headache may of course be a symptom of uremia, the recognition of which needs to be prompt. Headache due to uremia may be most pronounced in the back of the head and

neck, though it may be frontal. Dizziness is often present, and the patient is dull and apathetic. The headache of diabetes may precede the onset of diabetic coma, but it is rather an infrequent symptom.

Toxic Headaches. Among the causes of headache, chronic alcoholic poisoning should be borne in mind. The pain complained of is usually diffuse, dull in character, and most pronounced in the frontal region. It is usually most marked in the morning, and is increased by the additional drinking of alcohol. Associated with the headache are the other signs of alcoholism. Among the less frequent causes of headache, lead poisoning should be considered. Headache due to lead is prodromal to or associated with other cerebral symptoms, such as convulsions, delirium and stupor. Optic neuritis may also be present. In searching for the obscure cause of a headache, the abuse of tea, coffee, and tobacco must also be kept in mind. Care should be taken, however, not to confuse these headaches with migraine. Tobacco headaches occur irregularly and usually bear a clear relation to excessive smoking.

Headaches from Infection. Headaches associated with the various acute infectious diseases do not require special consideration; they are found as parts of well-known symptom groups.

Headaches symptomatic of affections of the special sense organs, such as the eye and the various viscera, are very important. The most frequent of these headaches is that due to eye-strain. Usually there is here an associated condition of nervous exhaustion, so that any unusual effort is followed by a headache. The eye-muscles, like other muscles, are readily fatigued in neurasthenia, and it is not surprising that the use of the eyes produces headache, especially when some refractive error such as astigmatism or hypermetropia necessitates a special effort at accommodation. Such a patient usually sleeps well, and rises in the morning free from headache. About the middle of the day, however, or after the eyes have been used for a few hours, the headache comes on, and frequently interrupts the patient's work. Rest relieves the pain. The latter may be either frontal or occipital in distribution, more frequently the latter.

In the treatment of eye-headaches, it is important of course that the eyes be corrected, but while this may relieve it may

fail to cure the headache. The underlying neurasthenia demands attention, and its efficient treatment may be the only means of effecting a permanent result.

Disease of the nasal chambers or sinuses may lead to headache referred to the brow, the temples, or the malar regions. In most of these cases the symptoms referable directly to the nose or sinuses are so clear as to establish the diagnosis. At times, however, the local symptoms are slight, and the cause of the headache may be overlooked unless especially sought for. In all obscure cases of headache, the nose and its accessory sinuses should be thoroughly examined.

Headaches are also produced by functional disturbances of the stomach and intestines. Such headaches are usually present when signs of indigestion are most marked; the pain is frequently relieved by vomiting. The headache is probably caused by the absorption of toxic materials from the stomach or intestine. The treatment is, of course, the treatment of the underlying digestive disturbance.

Disease of the uterus and ovaries is at times accompanied by headache. The pain is usually referred to the vertex, and may be temporarily relieved by pressure. The patient presents the well-marked symptoms of pelvic disease, and nearly always suffers from an associated symptomatic neurasthenia.

Headache Due to Anemia and Hyperemia of the Brain and Diseases of the Blood. A generation ago it was quite common to attribute headache to anemia and hyperemia of the brain. It is very doubtful, however, whether these conditions, when present, play any rôle. Thus in acute general anemia, *e.g.*, from hemorrhage, there is doubtless also an anemia of the brain, but the symptoms are dizziness, faintness, nausea, vomiting, tinnitus, failing vision and perhaps unconsciousness, with dilatation of the pupils; at no time does the patient complain of headache. Again, in chronic anemia, as from repeated hemorrhages, or in disease of the blood in which the hemoglobin is greatly reduced—*e.g.*, pernicious anemia—weakness, dizziness, apathy and somnolence may be prominent symptoms, but seldom does the patient complain of headache. On the other hand, cerebral hyperemia, though it may play a rôle, cannot be considered as an independent cause of headache. Thus, cerebral hyperemia probably occurs during febrile con-

ditions, but the associated headache is probably only in part due to this cause. Again, a plethoric person indulging in a full meal, and especially alcohol, may become flushed in the face, and it is reasonable to infer that the brain is also hyperemic. Such a patient may complain of throbbing in the temples, dizziness, fullness in the head and headache. Further, hyperemia, without doubt, occurs passively in venous obstruction, as in mitral stenosis, but headache, if present, is clearly secondary in its origin.

Clearly anemia and hyperemia of the brain play either no or a very doubtful rôle in the production of headaches; and it is significant that after neurasthenia became more fully understood these diagnoses were but rarely made.

MIGRAINE.

Migraine is an affection characterized by irregularly recurring attacks of headache, the pain being usually limited to or most marked on one side of the head; hence the name semi-crania. The pain is frequently accompanied by nausea and vomiting, various paresthesias and visual disturbances.

The disease rarely begins after 30 years of age. It may attack young children, but most frequently has its origin during the period of adolescence. Heredity appears to play a part in the etiology; it frequently occurs in the same family for two or more generations, and several members of the same generation may be affected. Excessive mental work, poor physical condition, and various reflex disturbances, as, for example, eye-strain, appear to have an influence in inducing attacks of migraine. Indiscretion in diet, the abuse of alcohol and tobacco, tea, and coffee, also favor attacks in those predisposed.

An apparent relation of migraine to epilepsy has been pointed out. It has been suggested that migraine is a sensory equivalent of epilepsy. However, when we consider that epilepsy is a condition, and not a disease entity, that many families present individuals afflicted with migraine but not with epilepsy, and we realize that the epilepsies are very varied in origin, the relation of migraine to epilepsy becomes very

doubtful. Even in individual instances, themselves very infrequent, the relation is at most only casual.

Patients are frequently aware for several hours that an attack is coming on. They may complain of weariness and exhaustion, or of heaviness, fullness or pressure in the head, sometimes of dizziness. Frequently the digestion is disturbed. Many patients preceding the attack note bright quivering lines or bright colors, visual scotomata, occupying a part of the visual field. At times, a hemianopsia is simulated. Some cases complain of tinnitus, while others present paresthesia, most frequently a numbness or pricking involving one side of the face, part of an extremity, or it may be the entire half of the body. At times, also, though infrequently, these symptoms may be accompanied by disturbances of motion, especially in face and tongue, so that the patient may not speak with his usual distinctness.

Vasomotor and pupillary disturbances may also be present; the face on the side affected may at first be flushed, and later pale; the pupil may be at first contracted, and later dilated. Nausea and vomiting not infrequently occur. Sometimes the pain is relieved by the vomiting; at other times it is accentuated. The patient is usually very sensitive to light and noises. Motion or jarring of the head commonly greatly increases the pain.

TREATMENT.

First, the general health of the patient should be brought to as high a level as possible. Great attention should be given to dietary and hygienic measures. Many of the patients are in general ill-health; and rest methods, either partial or complete, should be carried out whenever possible. (See page 583.) As in neurasthenia, the diet must be modified so as to reduce the starches and sugars very decidedly. The same is true of the red meats. The white meats, chicken and fish, eggs, milk, and the succulent vegetables are to be allowed freely. Milk is a very important article of diet, and severe cases often do well on rest and a diet limited to milk for a time. Further, it is highly nutritious, and at the same time favors elimination. Milk is of special value because it contains no nuclein, and therefore cannot lead to the formation of uric acid or other

leucomaines. Water is to be taken freely at all times. The skin should be kept active by warm sponge bathing. General massage is also employed with advantage.

Indigestion is to be guarded against as far as possible. Constipation should be corrected by simple laxatives, such as stimulate elimination, *e.g.,* sodium phosphate. At times cascara is useful. Enemas may occasionally be used, but should not be instituted as a routine procedure. Occasionally a brisk saline cathartic taken in the very beginning cuts short an attack. Intestinal antiseptics do little good.

Great benefit is often derived from the free and rather prolonged administrations of the salicylates. Ten grains (0.65 Gm.) of sodium salicylate together with 20 grains (1.30 Gm.) of sodium bromid, given well dissolved, three times daily after meals often influences the migraine decidedly. Aspirin when well tolerated is often of the greatest value. Salophen, more readily and easily tolerated, is not so efficient.

The treatment of the individual attack itself must also be considered. In severe cases, as soon as the prodromal symptoms make their appearance, a full dose of salts or laxative water should be administered. Later bromids, preferably ammonium bromid, should be given in a dose of 30 grains (1.9 Gm.). The patient should lie down and try to sleep, the room being darkened. In cases of less severity, or if the patient's engagements are such that he must if possible meet them, one of the coal tar products, antipyrin or phenacetin, 3, 5, or 10 grains (0.20, 0.30, or 0.65 Gm.), with or without ½ or 1 grain (0.032 or 0.065 Gm.) of caffein, may be given, often with the very best result. Sometimes 5 or 10 grains (0.32 or 0.65 Gm.) of aspirin answer equally well. Of especial service, however, the writers have found cannabis indica. The patient is instructed to take 1 drop (0.06 mil) of the fluidextract every half-hour until some relief is obtained. If 1 drop is insufficient, 2, 3 or more can be given. Thus the dose required to control an attack can readily be determined. Most patients are relieved by a small initial dose, but sometimes it is necessary to increase the dose to 5 or 10 drops (0.3 or 0.6 mil), or even more, to relieve the patient. The patient should be informed as to the physiologic action of the drug in full doses—the dizziness, confusion and disturbances in the sense of time that may pos-

sibly make their appearance. These symptoms, alarming to the patient, are of very little significance. Cannabis indica has the great advantage of never causing depression, and of never leading to a drug-habit. Sometimes it fails, due usually to an inefficient preparation. In such case we may be obliged to have recourse to gelsemium. This should be cautiously administered in a similar manner. It is, however, less efficient, and the fact that in full doses it is both a nervous and cardiovascular depressant should be borne in mind.

The drug which above all others has the power to relieve pain is, of course, morphin; but the fact that in a disease with recurrent attacks, such as migraine, it leads sooner or later to the formation of the morphin habit, makes it inadmissible. Physiologic methods of living, proper regulation of the diet, relief from excessive work and nerve-strain, together with the general up-building of the patient, and combined with the thorough use of salicylates and bromids, will bring the attacks under measurable control, so that recourse to analgesic remedies will become less necessary.

VERTIGO.

Vertigo is a symptom in which the patient is conscious of a disturbance of his equilibrium; there is a subjective sense of movement, or of actual movement, accompanied by a more or less marked disturbance in, or even loss of, the sense of space relations. This disturbance the mind translates into an illusion of turning or rotation. This movement may be referred by the patient to his own person, or may be projected by him to the external world; *i.e.,* the patient may experience a sensation as though he himself were turning, or as though the objects about him were turning; hence the term vertigo, derived from the verb *vertere,* to turn. The terms giddiness and dizziness are both commonly applied to the less marked conditions of vertigo, or to forms that are so slight as to be almost if not entirely subjective.

Vertigo is a symptom which is found in a great variety of conditions; only at times does it appear as an independent clinical affection; more frequently it is purely symptomatic. It may be a symptom of a general affection such as neuras-

thenia or hysteria. It may be met with in toxic states, *e.g.*, those due to alcohol, tobacco and coffee; in uremia and in the early periods of the infectious fevers. It may be met with in disturbances of the digestive tract and of the circulatory apparatus. It may be met with in disturbances of special sense organs, such as the eye, and especially the ear. In the eye it may be due to a refractive error or a loss of muscle balance. In the ear it may be due to disturbances of the semicircular canals or of other portions of the auditory apparatus. Again, it may be due to organic brain disease, more especially of the cerebellum or its peduncles. Finally, cases are met with in which no cause can be discovered.

That vertigo may be excited artificially is known to every child who tries the experiment of turning rapidly around a number of times in succession. The vertiginous sensations of sea-sickness have doubtless an origin in a similar disturbance of the sense of space relations. Again, vertigo may be excited by douching the ear with water, or by the passage of a galvanic current through the ears. This so-called galvanic vertigo is remarkable for the fact that the person experimented upon tends to fall toward the anode on the closure of the current, and toward the cathode on the opening of the current.

In every case of vertigo, the important matter is the diagnosis as to cause. As may be inferred, this is by no means always possible. If the vertigo be clearly objective, and if the signs of organic brain disease be present, much information may be gained as to the site of the lesion by the Bárány tests. This depends upon the fact that in vertigo induced by rotation, or by douching the ear, the symptoms are accompanied by nystagmus. Nystagmus, however, can only take place provided that the inferior longitudinal fasciculus, which connects the nucleus of Deiters with the nuclei of the eye-muscles, is open and intact. It is very clear that when the associated nystagmus is absent or deficient, the lesion must lie in this tract, or at least that it is cerebral and not cerebellar. On the other hand, if it be not interfered with, and there are other signs of organic brain disease, the inference is obvious that the lesion is cerebellar or peduncular. These inferences are justified provided of course that disease of the semicircular canals itself has been excluded. A special revolving chair and a

highly specialized technique has been devised by Bárány, which, in trained hands, yields most interesting and detailed information.

The affections of the ear which may be attended by vertigo are most varied. The disturbance may have its origin in the external, the middle, or the internal ear. Commonly, more or less decided impairment of hearing is present. Usually this impairment is accompanied by a diminution of bone conduction, and with especial frequency do we find tinnitus aurium. However, neither impairment of hearing nor tinnitus is necessarily present. The symptoms in the majority of cases point to disease of the labyrinth; disease of the meatus or middle ear should always be excluded, and the Bárány tests should whenever practicable be made. In the form of vertigo described by Menière, the cause appears to have been hemorrhage into the labyrinth. Menière's disease in the experience of the writer is quite rare. Frankl-Hochwart was able to find but twenty-seven cases in the literature. The symptom group is somewhat as follows: There is an apoplectiform onset; the patient may fall to the ground; there is present severe nausea, vomiting, deafness and tinnitus. Curiously, the impairment of hearing is usually bilateral. Gradually the nausea and vomiting subside, and, little by little, the vertigo lessens, and may altogether disappear. More or less marked impairment of hearing, however, as a rule, remains. The diagnosis of Menière's disease is to be based upon the suddenness of the attack, the deafness, the tinnitus, the intense character of the vertigo, and especially upon the results of the Bárány tests.

Ocular vertigo is very rare. It is at times associated with double vision; at others with an erroneous projection of the visual field. It is seldom intense, and the cause is usually revealed by the ophthalmologic examination.

The vertigo associated with disturbances of the stomach is so closely associated with the taking of food and with the symptoms of indigestion, that the diagnosis is, as a rule, very readily made. This is also true of the vertigo associated with constipation.

Disease of the cardio-vascular apparatus is not infrequently accompanied by vertigo. In cases of heart disease it must of course be differentiated from transient attacks of cardiac

weakness. It is also important to bear in mind that vertigo may be an accompaniment of sclerosis of the cerebral vessels, and possibly of general arteriosclerosis.

The determination of the cause of vertigo depends, of course, upon the detailed study of each individual case. It should be further borne in mind that the milder forms of vertigo, especially those to which the patient applies the term dizziness or giddiness, are subjective, and are relatively unimportant. Quite commonly they are part of the symptom-group of neurasthenia, and rapidly disappear with treatment. General principles must also guide the practitioner in the treatment of vertigo when the latter is dependent upon visceral disease; this is also the case when no special cause can be discovered. Among other things, also, the possibility of a toxemia should be considered.

When the vertigo is labyrinthine and persistent, purgatives, sweating, counterirritation back of the ear, absolute rest in bed, the iodids, bromids, and perhaps other sedatives may be employed. These and similar measures are also justifiable when no cause for the vertigo can be discovered. It may be worth while to add that Babinski has in some cases practised lumbar puncture with benefit. In very desperate and severe cases, the attempt appears to be justifiable to secure relief by extirpation of the semicircular canals. Lake and Milligan report such cases.

EPILEPSY.

Epilepsy is a symptom-group of multiple origin characterized by irregularly recurring attacks in which loss of consciousness is the dominant feature. The attacks may or may not be attended by convulsions.

Petit mal, or the mild form, is characterized merely by a momentary loss of consciousness, while *grand mal,* or the major form, presents, in addition to the loss of consciousness, also convulsions. The attacks are frequently ushered in by premonitory symptoms, usually sensory, though sometimes motor. They are spoken of as the aura, or signal, symptom. It is frequently described as a numbness or tingling, or other strange sensation arising in an extremity, in the epigastrium or elsewhere, and spreading upward to the head, when con-

sciousness is lost and the attack supervenes. The aura may also arise in one of the special senses; thus the attack may begin by ringing in the ears, bright flashes of light and colors, or by strange tastes and smells. Not infrequently the attack is ushered in by a cry. The patient loses consciousness, falls to the ground, passes into a tonic spasm involving in the generalized form the muscles of the limbs, trunk and head. This tonic spasm is of very brief duration, and is immediately succeeded by active clonic movements, usually quite rapid, though not very large in extent. The duration of the attack usually covers a few minutes only. Gradually the violence of the convulsion moderates, the patient becomes quiet, and consciousness returns. The patient seems somewhat heavy and stupid, may complain of headache, may vomit, and usually falls asleep. Not infrequently the tongue is bitten during the attack, while the urine, and at times, though much less frequently, the bowels may be voided.

Sometimes instead of having a convulsion, the patient may become suddenly and actively confused or delirious. Such states may last several hours, or even a number of days, and during their continuance he may be destructive and very violent. Fatal assaults may even be committed. At other times comparatively mild states of automatism alone are present.

Epilepsy, or the epilepsies, as we should properly speak of them, are of very varied value and character. Thus, in a notable percentage of cases we meet with neuropathic family histories, and in a smaller number a history of a collateral or a direct heredity. In others, again, there is a history of alcoholism or of syphilis in the ancestry, and the inference is unavoidable that in a large number of epileptics, there has been a primary, a basic impairment of the germ plasm. This impairment may be general in character, the result of various intoxications and infections in the ancestry; or the impairment may be special in character, and may result in the special transmission of epilepsy.

Concerning the evidence of the direct production of epilepsy in the individual himself by intoxications and infections, the evidence is overwhelming. This is notably true of alcohol. Again, the convulsions which at times accompany or usher in

the acute infectious diseases of childhood are to be regarded merely as epiphenomena of the infectious process, and are to be explained by a direct toxic action on the brain cortex. Such convulsive seizures generally disappear with the infection, but unfortunately they now and then persist as established epilepsies. Sometimes there is an interval of months or years during which the convulsions are absent, and after which they reappear. It is probable that in such cases an encephalitis, perhaps limited in area, has occurred during the attack of the infectious disease, and that this has been followed by sclerotic changes, the latter being then sufficient to act as the starting point for epileptic attacks.

Another factor in the production of epilepsy is trauma. Epilepsy following injury to the brain is generally Jacksonian in type, but it is not improbable that changes may supervene in the traumas of childhood which may later give rise to the picture of a generalized epilepsy.

When we study the etiology of epilepsy we are impressed with the fact that epilepsy is not a definite clinical entity; that under this name are included many symptom-groups, which differ widely as to their origin. The pathologic findings are of significance, and in keeping with this view. Many years ago, one of us placed on record anatomic studies of twelve epileptic brains, all of which revealed more or less marked anomalies of the convolutions and fissures. These findings were to be interpreted as phenomena of arrest and deviation. A similar interpretation is to be placed on the sclerosis of the cornu ammonis, so much insisted on by the earlier writers. Developmental arrest and deviation have the same significance as the asymmetries and malformations of the skull. Microscopic studies of the brain have sometimes revealed atrophic changes in the cortical cells, and sometimes a proliferation of the glia. Macroscopic studies have shown thickening of the skull and membranes, and the adhesion of the latter to the skull or brain. The factor of most significance, however, is that none of these findings are constant.

Some epileptics present symptoms suggestive of involvement of the internal secretions, but anomalies in the development of the glands of internal secretions may well accompany general arrest and deviation. No special internal symptom-

group is presented. In quite a number of epileptics, Dercum demonstrated a number of years ago, by Röntgen-ray examination, enlargement and distortion of the pituitary fossa due apparently to disease of the hypophysis.

TREATMENT.

The first consideration of treatment is that the patient lead as physiologic a life as possible. There should be little or no mental or physical strain. The patient should live close to nature on farm or in camp. This is the aim and object in the various epileptic colonies. Under such circumstances many patients are improved in general health, and the frequency of attacks lessened. Undoubtedly, benefit arises from the increased oxidation of waste and toxic products, brought about by the life in the open, and by the increased physiologic efficiency.

The diet should be so modified that in a patient already toxic as little strain as possible be placed on the liver, thyroid, kidney and other defensive glands. Very little meat, and especially red meats, should be eaten. Carbohydrates should be diminished. Large amounts hamper the oxidation of the tissues, which, in a patient suffering from autointoxication, should be maintained at as high a level as possible. Succulent vegetables and milk can be given freely. Stimulants such as alcohol, tobacco, tea and coffee are to be excluded. The various avenues of elimination must be kept freely open. If constipation is troublesome, salines, laxative waters or cascara sagrada may be given. Thorough irrigation of the large intestine once or twice weekly with plain water benefits many cases.

Water should be taken freely between meals, to promote the action of the kidneys. Daily sponge bathing or a lukewarm tub bath will stimulate the action of the skin. Cold or hot baths are not indicated.

No matter how carefully the life of the patient is regulated, we must in many cases resort to medicines. Mild cases, with attacks at infrequent intervals, may do well on proper hygiene and diet, but those having frequent attacks do better under medication. The usefulness of the various bromid salts gives

them the first claim to our attention. In the administration of the bromid we should remember that the sodium or ammonium salts are less depressing than the potassium salt. Strontium bromid in the experience of the writers offers no special advantage. It is important, furthermore, to bear in mind the procedure introduced by Richet and Toulouse, that is, to reduce the amount of the sodium chlorid in the food of the patient to a minimum. The sodium bromid appears in a measure to substitute the sodium chlorid in the tissues. It is to a great extent retained in the economy and is efficient in a much smaller dose.

Chloral hydrate may be combined with bromids for a short time to control severe groups of seizures, but it should never be continued long. Occasionally small doses of belladonna or of hyoscyamus increase the efficiency of the bromids, but they also are not suited for continued administration. Fowler's solution in small doses may be given with the bromids to lessen the tendency to acne. Antipyrin also given with bromids may re-enforce the action of the latter. Among the more recent remedies found to be of value in controlling the seizures are luminal and luminal sodium. Given in 1- to 3-grain (0.06 to 0.19 Gm.) doses three times daily they are very efficacious, and are unaccompanied by any unfavorable effects. The remedy may apparently be continued for a long time.

If the epilepsy be purely nocturnal, a 3-grain (0.19 Gm.) dose of luminal or luminal sodium, or sulphonal, 5 to 10 grains (0.32 to 0.65 Gm.), proves very satisfactory in preventing the seizures.

At times the administration of small doses of thyroid does good. It is probable that the thyroid extract acts by stimulating the chain of glands of internal secretion generally, and thus increasing metabolism. Probably the oxidation of waste substances is favored by this means.

INFANTILE CONVULSIONS.

Infants and young children are more liable to convulsive seizures than older children or adults. This is possibly due to the more irritable condition of the nerve centers, and a comparatively feeble inhibition.

Clinically infantile convulsions separate themselves into those which occur immediately or shortly after birth, and those which occur after the lapse of several months, or within the first two or three years. The convulsions of the new-born may be due to traumata of the brain occurring during difficult labor, *e.g.*, from prolonged compression of the head, or other conditions involving delay and instrumental interference. The most common lesion is meningeal hemorrhage. The convulsions may be slight and brief, or they may be severe and prolonged. Further, they may be accompanied by distinct localizing signs.

If indications are at all clear, such cases should be treated surgically, the skull opened and the clots evacuated. How much can be accomplished has been shown by Cushing. How urgent surgical interference is, is shown by the disastrous results of meningeal hemorrhage. Mental arrest, diplegia, hemiplegia, and epilepsy are the common sequelæ.

Convulsions such as are due to organic lesions in the brain and meninges are rare in the first few months of life. Subsequently convulsions may make their appearance, *e.g.*, during the period of dentition, and are then generally due to some gastro-intestinal disturbance. Quite commonly the explanation is to be sought in an attack of indigestion, the result of overfeeding, or of the giving of unsuitable food. The convulsion may come on while there is still undigested food in the stomach, and in such case the attack may be relieved by vomiting. More frequently, however, the convulsion does not come on until several hours have elapsed; intestinal indigestion may then be the direct cause of the convulsion. The formation of irritant and toxic materials appears also to play a rôle. Chronic gastro-intestinal disturbances, *e.g.*, gastro-enteritis and intestinal infection, it should be added, are less frequently accompanied by convulsions than are acute disturbances.

Among other causes of convulsions in infancy and childhood are the infectious diseases. Here the special cause at work is apparently the action on the cortex by the bacteria and their toxins. Convulsions, too, occur more frequently in children of neuropathic make-up and heredity, and it is not impossible in such cases various causes of peripheral irritation may play a rôle, though the evidence is usually not conclusive. The pos-

sibility of intestinal parasites, and, lastly, of uremia should be borne in mind.

Infantile convulsions in a general way resemble epileptic seizures. Frequently also there are prodromal signs. The child is apt to be restless. Occasionally the local twitching of a muscle or of an extremity may be noted; often, too, there is gritting of the teeth. The pulse rate is usually decidedly increased, and there may also be a rise of temperature. The latter of course suggests a gastro-intestinal or other infection.

The convulsion generally comes on suddenly. Distinct shocks make their appearance in the extremities, and the convulsion follows. The eyes may turn upward, may deviate to one side, or may rapidly twitch to and fro; or a transient or intermittent strabismus may be noted. The pupils vary, though they are usually contracted, the more so the more violent the seizure. Usually, as in epilepsy, the attack begins as a tonic spasm, quickly followed by clonic movements; but it may be clonic throughout. The urine and bowels are usually not evacuated, but this may occur. The child is, as a rule, quite unconscious, and, if the convulsion be prolonged, it becomes cyanotic owing to interference with respiration. It may cry out as the attack comes on or subsides. Usually there is no cry during the attack. The convulsive seizures may subside, may be repeated, or may terminate fatally. Increase of pulse-rate is present during the intervals. Fever also may be present.

Death may ensue during a convulsion, either from exhaustion or from the toxins of the disease producing the convulsion. Not infrequently it is preceded by a rapid and high rise in temperature.

TREATMENT.

The treatment of infantile convulsions must be directed if possible alike to the cause and to the convulsion itself. If the attack owes its origin to a digestive disturbance, we should, according to circumstances, encourage vomiting, practise free lavage of the bowel, using, if there be fever, cold or iced water. Free evacuations should, if possible, be secured by small doses of calomel, followed by castor oil. Other things equal, the child should be immersed in a warm bath, and cold applied to

the head. Bromid, bromid and chloral, or bromid and antipyrin may be given by the mouth, or perhaps better by the bowel. Caution and judgment must of course be exercised, both as regards the size of the doses and their repetition. If the convulsions are very severe and persistent, it is perfectly justifiable to make a cautious trial of a few drops of chloroform inhalation. Finally, we believe it to be perfectly proper also, in such cases, to resort to spinal puncture.

In former years difficult dentition was popularly believed to be a cause of convulsions. It cannot do any harm, of course, in a given case to freely lance the gums. In cases in which the convulsions are part of the invasion of one of the exanthemata, the management of the case is of course that of the infection. The convulsion itself, however, may demand immediate attention.

PUERPERAL CONVULSIONS.

Convulsive seizures may make their appearance during pregnancy, usually during the latter part of gestation, during childbirth, or during the puerperal period. They occur most frequently in young primiparæ. They may be renal in origin, but it frequently happens that the urine is entirely negative to examination, both as to albumin and casts; at most only a trace of albumin may be found. On the other hand, women with well recognized Bright's disease may successfully pass through both pregnancy and childbirth. An adequate explanation of puerperal convulsions has not yet presented itself. Of course, a toxic cause seems necessarily to be present. It may be that a special disturbance of metabolism is present, together perhaps with renal insufficiency. There are good reasons for believing that neither the fetus nor the placenta is the cause of the eclampsia; a convulsion may occur after the uterus is empty, the fetus and placenta having been expelled; or it may even occur with a hydatiform mole.

Prodromal symptoms, if present, consist of headache, visual disturbances, epigastric distress, restlessness and general discomfort. The attacks greatly resemble epilepsy. Further they may be very violent and prolonged, as in status epilepticus.

TREATMENT.

If patient is in labor, it seems best to hasten the latter as much as possible. On the other hand, if labor has not begun, it is the consensus of opinion not to induce labor, nor to empty the uterus by surgical procedure, such as section, but to endeavor to control the seizures. If the latter are severe, this may be accomplished by the cautious use of an anesthetic, such as ether or chloroform. Bromids, chloral, morphin, may also be given in full doses, either separately or in combination, as may seem wise. In order to combat the toxicity which is undoubtedly present, it may be well to practise venesection, and to follow the latter by a simple saline solution intravenously. Hypodermoclysis may be practised instead, but its action is less direct and slower. It is in keeping further with modern procedures to add sodium bicarbonate to either the intravenous or the hypodermoclysis. Alkalies can also be given by the bowel.

Diaphoresis should be encouraged. The hot pack may prove very serviceable. Salines and liquids should be freely administered by the mouth.

CHOREA.

The symptom-group chorea presents itself in various forms; *e.g.,* as the chorea of childhood, chorea minor, Sydenham's chorea or St. Vitus' dance, as it is variously termed; also Huntingdon's chorea and so-called electric chorea.

Sydenham's chorea is an affection of childhood characterized by irregular, involuntary, incoördinate movements. It usually appears between the seventh and the thirteenth years, and is more common in girls than in boys in the proportion of 3 to 1. Occurring after the fifteenth year, it is generally found in the female sex. Again, it may occur in youth or in adult life, though it is very rare in the latter period. Finally, it is among the rare sequelæ of scarlet fever, measles or typhoid, and it is noteworthy that the prognosis in such cases is not so favorable as in ordinary chorea. Occasionally chorea is noted during pregnancy. Here it usually occurs in primiparæ, and during the early months. It is also rarely met with in old age, but we may question the correctness of classifying senile

chorea with the chorea of Sydenham. In the chorea of childhood there is frequently a prodromal period of pains in the joints. A history of swelling in the joints cannot be elicited; nevertheless the fact points to an infectious process. Not infrequently, also, an endocarditis is noted. The consensus of opinion favors the view of a microbic infection, though a specific germ has not been isolated. Possibly infections finding their way through the nose, throat, tonsils or middle ears play here a rôle.

The patient may in the beginning be dull, peevish, irritable, perhaps a trifle awkward and restless. Soon irregular muscular movements begin, generally in one hand, extending to the arm and face, or may spread over the entire half of the body. Occasionally it is limited to one-half of the body, and is then spoken of as hemichorea. In the larger number of cases, however, the opposite side also becomes involved. The movements do not consist of sudden twitchings or spasmodic movements, but are slower, and to some extent simulate voluntary movements. The movements are generally most marked in the extremities and in the face, but they may involve the eye muscles, the muscles of speech, and even of deglutition.

Quite commonly the affected muscles are somewhat weak. The electric reactions, however, of both nerves and muscles remain normal. The reflexes are usually unchanged, and there is no involvement of the sphincters. Choreic movements cease during sleep. They may, however, be so pronounced as to seriously interfere with sleep. Again they may be so violent and so widely diffused as to make it impossible for the patient either to stand or to walk. Mentally the patient is irritable and emotional, or dull and apathetic. In very severe cases he may become confused and even delirious.

The duration of the chorea of childhood is generally from six to twelve weeks. The prognosis is on the whole very favorable; a small percentage (only 3 or 4) die of exhaustion, or of cardiac or other complication. The more prolonged cases are apt to be among older patients, and are more often complicated by endocarditis. It is important to bear in mind that one attack does not confer immunity, but the affection is prone to recur. At least one-fourth of the cases suffer from two, three and often more attacks.

TREATMENT.

Rest is the first principle of treatment. The child should be taken from school, and, if the disease becomes at all severe, rest in bed should be instituted. In the milder cases the patient may be permitted to remain up and about, but even here the hours spent in bed should be increased. The diet should be liberal and nutritious. Milk should be given freely, both with and between meals.

Arsenic is regarded as the most reliable medicinal agent. It should be given in very small doses at first, preferably 1 drop (0.06 mil) of Fowler's solution three times daily. This may be gradually increased, by 1 drop (0.06 mil) daily until 3 or possibly 4 drops (0.18 to 0.24 mil) are given three times daily. It should then be discontinued for a few days, or the dose diminished drop by drop until the original dose is reached, and then another course given. The physician should, of course, be alert, and discontinue the medicine altogether if puffiness about the eyes, or gastric or intestinal disturbance be noted. Arsenic is, of course, not necessary to the successful treatment of chorea; and unless the physician has confidence in the mother, or is reasonably sure that the patient will be brought to see him at reasonably frequent intervals, he should not prescribe the remedy at all. Every now and then serious chronic poisoning results from small doses too long continued, and neuritis, wasting, and palsy may be the result. Iron is a much safer tonic. Occasionally, the salicylates are indicated, and prove serviceable. In very severe cases, in which the movements are violent and continuous, small doses of trional, veronal, or luminal may be given. The bromids also may be tried. Chloral is rarely indicated.

Children who have suffered from chorea should receive close personal attention; the throat, tonsils, nose, gastro-intestinal tract and other possible avenues of infection should be carefully studied.

HUNTINGDON'S CHOREA.

Huntingdon's chorea is a hereditary form of chorea, which occurs most frequently between the ages of 35 and 40 years. It rarely begins before 30 or after 45.

The disease is distinctly hereditary; it is transmitted directly from one generation to the next. Huntingdon in his original paper stated that his father and grandfather, who had practised medicine in the eastern end of Long Island for years, had known certain families in which this disease had existed for generations. In these families there were usually, also, members who were unaffected; the descendants of the latter commonly escaped. Both sexes are equally liable.

The disease comes on gradually. It generally begins in the face and upper extremities; the movements at first are slight. Later they become more general and more pronounced. Not only the limbs, but the muscles of the trunk and neck, and even of the throat, also become involved. The movements are involuntary, incoördinate, and almost uninterrupted. The involvement of the face, neck and throat muscles leads to difficulty in speech, and at times of swallowing. The patient makes grimaces, gesticulates, walks, it may be, with legs wide apart, tottering or tripping, now very slowly, now faster, or he may stop abruptly altogether. In his motions, the patient may suggest the behavior of a clown.

The movements are wider in range and more extensive than in ordinary or Sydenham's chorea. Attempt to control them or emotional excitement makes them worse. The strength of the muscles does not seem lessened. Sensation is normal. The reflexes are as a rule increased. Mental symptoms are commonly added to the picture. The patient is depressed; sometimes he entertains persecutory ideas. Later mental failure becomes evident. The disease is essentially degenerative. It is very slowly progressive and incurable. As the years pass by, the patients gradually lose strength and are finally confined to bed. It is an affection of a very slow course, a duration of ten to thirty years not being uncommon.

We possess no definite knowledge of its pathology. Various anatomic changes have been found in the nervous system, but the connection of these with the symptoms is not clear. Among these changes are disseminated foci of sclerosis, diffuse meningoencephalitis, atrophic changes in the cortex, proliferation of neuroglia, infiltration of the cortex, with glia cells, atrophic changes in the central convolutions, or it may be in other portions of the brain.

TREATMENT.

The treatment is purely symptomatic. Practically we are restricted to simple hygienic care. Rest methods and other physiologic procedures, so useful in functional nervous diseases, fail here altogether. Gentle gymnastic exercises have been recommended, and are perhaps useful in encouraging and occupying the patient. At times the movements become very pronounced, and in such case warm baths and sedatives should be resorted to. Occasionally, too, the mental symptoms become so severe as to necessitate the commitment of the patient to an institution.

ELECTRIC CHOREA.

Under this name are described various affections of unknown origin, and a legitimate doubt arises as to whether the term chorea is not improperly applied to them. It is not improbable that many cases of so-called electric chorea really belong to the category of hysteria. The term has been applied to cases in which the movements occur with great suddenness. Henoch separated from the ordinary form of chorea a disease picture, which he called electric chorea, and in which the muscular twitchings follow one another with lightning rapidity, differing in this respect from Sydenham's chorea. The twitchings affect generally the muscles of the shoulder and neck. A similar form has been described by Hirsch. It is probable that we have to do here with a hysterical myoclonia. In the form of electric chorea described by Bergeron, we have an affection in which sudden spasms make their appearance in children from 7 to 14 years of age, children usually of delicate and anemic appearance. The spasms affect the muscles of the back of the neck, shoulders and arms, and shake the entire body. Occasionally, however, one extremity only is affected. Attempts at control usually aggravate the spasms. The affection likewise strongly suggests hysteria. The prognosis is uniformly favorable, the conditions yielding to simple physiologic methods and tonics.

Dubini describes an affection occurring in northern Italy, which begins with pain in the neck and back; soon lightning-like contractions make their appearance in one-half of the

body, namely, in the face, arm and leg. Subsequently the opposite side of the body becomes involved. Occasionally epileptiform attacks and paralytic phenomena are added. Pain and fever are also present. Later, widely diffused palsies, with wasting of muscles and change in the electric reactions, follow. The disease generally terminates in death; recovery is rare. Heart failure and coma terminate the picture. It is probably an infectious disease.

TIC.

In the consideration of psychasthenia, in the previous pages, defects of inhibition were pointed out which express themselves in various movements. These movements frequently become pronounced, and are spoken of as tic, or tic convulsif. Quite commonly they suggest some voluntary or automatic gestures.

The milder form of tic occurs most frequently about the face. Very frequently it consists of winking, or of winking associated with other movements. At other times, the mouth and lips are involved, and grimaces, sniffling, sudden protrusion of the tongue, and other bizarre movements result. Sometimes it is the neck and shoulder; the patient may suddenly and repeatedly bow, shrug his shoulders, nod, turn or throw back his head, his action resembling some voluntary movement. The arms may be involved, the hand being carried to the head, the face or the beard. Similarly, the lower extremities may be affected, though to a less extent. The patient may suddenly rise from his chair, take a step or two, turn about or perform some other curious movement. At the moment of action the casual observer receives an impression as though the movement were made by design. At the same time the patient often emits sounds or exclamations; sometimes phrases or parts of phrases, the latter being thrown or interlarded in a senseless way, into the speech of the patient. Not infrequently the expressions are obscene and profane.

The phenomena recur at irregular intervals. By mental concentration, the movements may be controlled for a time; but frequently, after a too insistent repression, they recur with increased force. They are made worse also by excitement. In

addition to the movements, the patient may also suffer from phobias and obsessions. For a detailed consideration of the latter, the reader is referred to the section on psychasthenia.

The *treatment* is that of psychasthenia; rest, full feeding, and hygienic living. Light hydrotherapy and medical gymnastics are useful. Psychotherapy also is of value. (See p. 605.)

HABIT SPASM.

Closely allied, if not identical, with tic convulsif is a condition described by S. Weir Mitchell as habit chorea, and by Gowers as habit spasm. It is manifested by slight spasmodic movements of small groups of muscles, which result in winking or twitching of the mouth or other transient and slight grimaces. The movements have a semi-voluntary aspect. The affection usually occurs between the ages of 6 and 14 years, and is apt to subside as the child grows older. In rare cases slight movements continue during youth, and even throughout adult life. It is the general rule, however, that cases in which mild facial twitchings are present are likely to get well. Occasionally the movements are extensive, involving the trunk and limbs and then constitute true tic; in such a case, of course, they are likely to persist.

In *treatment* everything should be done to raise the level of the general health of the patient. An extensive consideration of treatment is here unnecessary. Rest, full feeding, proper hygiene, and the giving of tonics are important.

The patient must be examined for all peripheral sources of irritation, and these when discovered should be remedied. The eyes should be examined, and refracted if necessary. Any disease of the conjunctivæ should be treated. Similar attention should be given to the mouth, nose and throat. The teeth should be put in good condition; tonsils and adenoids removed if necessary; if an adherent prepuce is present, its removal is advised. The stools should be examined for worms. Very commonly, however, no source of peripheral irritation can be found.

LOCALIZED MYOSPASMS.

Myospasms affecting a muscle or group of muscles, either tonic or clonic may affect the muscles of almost any portion of the body. More frequently, however, special muscle groups or those representing special nerve distribution are involved.

Facial Spasm. Clonic facial spasm, or painless tic, the form most commonly met with, consists of an irregularly recurring contraction of the muscles supplied by the facial nerve. The contraction may involve all the facial muscles, or may be limited to certain groups. In the diffuse form the symptoms are limited to one-half of the face, though it occasionally happens that certain movements, such as winking, are bilateral. As a rule, the spasm begins in one group of muscles and spreads to the others; there is, however, no regularity in the order of sequence. Thus the mouth may be suddenly drawn up, the alæ of the nose twitch, the eyes blink, and then the whole side of the face becomes involved. The entire paroxysm usually lasts for a fraction of a second or for a few seconds only. At times, though infrequently, it lasts for a minute or even longer. In rare cases the spasm consists of a single muscular contraction; more frequently it is made up of a number of clonic movements which quickly increase in rapidity until a maximum is reached, when they again become slower and gradually die away. Sometimes both the onset and cessation of the spasm are abrupt. Following the spasm there ensues an interval, varying in different cases, in which the face is quiet, or almost so. Sometimes this interval lasts many minutes, and in mild cases the spasm may occur only occasionally in the course of the day. In other cases, the paroxysms occur with such frequency as almost to simulate a tonic spasm. In other cases, again, in which a decided pause is present between the spasms, minute local and isolated twitchings may occur during the interval. As a rule, the contraction of the zygomatic muscles and the elevators of the angles of the mouth and nose predominate over the contractions of the other muscles.

Next in frequency the spasm affects the orbicularis palpebrarum, and least frequently the depressor of the angle of the mouth. The occipito-frontalis, the muscles of the ear, the

muscles of the palate and the platysma are very rarely involved. However, no muscle of the facial supply is exempt. Occasionally, too, the spasm is not limited to the facial supply alone, but radiates into other nerve territories. In such cases it may involve the masseters and temporalis, or it may spread to the muscles of the neck, and even to the muscles of the arms and shoulders. No weakness can be detected in the affected muscles, nor is there any change in the electrical reactions. Further facial spasm is at times bilateral. The zygomatics of both sides may in such case be involved, and at short intervals a grin or smile passes over the patient's face. At other times the corrugators are affected, the patient suddenly frowning without cause. Again, the involvement of the orbicularis palpebrarum may be so slight as to cause a barely perceptible twitching of the eyelids, or it may be so pronounced that the contraction may last for several seconds, or even many minutes, or it may be so severe as to be practically continuous, and thus make the patient, to all intents and purposes, blind. The eyes may be so firmly closed during a spasm that no effort of the patient's will can open them. Curiously enough, blepharospasm can, at times, be relieved by pressure of the finger on certain points; for instance, over the supra-orbital notch or over the supra-orbital nerves upon the brow. Now and then such points are found on the infra-orbital branch of the fifth nerve, and sometimes upon areas that bear no relation whatever to nerve distribution. Thus they have been found on the back of the neck, on the shoulder, in the axilla, and on the arm as low down as the wrist. Pressure-points may be met with not only in blepharospasm, but also in generalized facial spasm. The point to be considered in these curious cases is whether or not hysteria plays a rôle.

TREATMENT.

The mouth, teeth, eyes and nose should be carefully examined. The fifth nerve should be thoroughly explored throughout its various divisions, and this result proving negative, the entire body should be examined, and, especially in children, the intestinal tract must not be forgotten.

Quite commonly nothing is found to which the affection can be attributed, and we are forced to treat the latter symp-

tomatically. Among the measures that have been adopted are counterirritation by blistering, or by the actual cautery over a small area over the cervical spine or back of the ear. Freezing of the face with a volatile spray may be tried, but neither counterirritation nor freezing gives very decided results. The results of the constant galvanic electric current and of other electric treatment are so poor as to hardly make them worth the trial.

Surgical treatment is of more or less benefit, though as a rule for a limited time only. Stretching of the facial nerve is very efficient, but as soon as the ensuing paralysis disappears the spasm reasserts itself. During the interval, however, the patient is relieved for many weeks, and even months. Section of the facial nerve, of course, yields a similar result, and the relief is naturally of longer duration, but when union of the nerve takes place the spasm returns.

Deep alcoholic and osmic acid injections may be tried. These often give decided results, which are usually more persistent than those obtained by stretching, while the resulting weakness of the nerve is not so serious as that resulting from section. Various drugs have been employed internally, but without decided benefit. In very severe cases of blepharospasm, and, indeed, all other forms of facial spasm, there is but one drug which has a marked effect, and this is morphin. When used internally, or, better still, when injected, hypodermically near the exit of the nerve, it markedly lessens the spasm. However, morphin is rarely a drug one is justified in using in such cases, the cure being worse than the disease. Deep alcohol injections are much to be preferred.

The general hygiene of the patient should receive care, as in other functional nervous diseases, and the reader is referred to the methods employed in the treatment of neurasthenia and hysteria. We should bear in mind that along with facial spasm, the patient often presents the signs of more or less marked deterioration of nervous health.

Tonic Facial Spasm. In a given number of cases we meet with tonic spasm, or contraction of the face. This is most frequently met with as an after-result of Bell's palsy; the muscles having been paralyzed for a long time, secondary contracture supervenes, just as it does in the muscles of the arms and legs

in hemiplegia. Very rarely tonic facial spasm is said to follow exposure to cold, and also to be an accompaniment of hysteria. The writers have never observed this. The treatment of tonic facial spasm or contracture is, as a rule, ineffectual, though sometimes vigorous facial massage, with stretching of the contracted muscles, is beneficial. The constant galvanic current also may be employed.

SPASMODIC TORTICOLLIS.

Spasmodic torticollis, also termed spinal accessory spasm, consists of a spasm of certain muscles by which the head and neck are rotated to one side, or from side to side. The spasm involves either one or more muscles, especially the sternocleidomastoid and the trapezius, but also the splenius, the scaleni, and the deeper rotators of the head and neck. Because of the relatively large size of the sternocleidomastoid and trapezius, the movement is largely influenced by their action. However, that rotatory spasm may occur independently of these muscles is shown by cases in which the spinal accessory nerve has been resected, the movement persisting in spite of the resulting paralysis.

Spasmodic torticollis is quite characteristic. Every few seconds or every minute the head is forcibly drawn to one side, the mastoid region depressed, the chin elevated and turned toward the opposite shoulder. There are many modifications of this movement, depending on the action of the muscles involved. It should be remembered too that the affection is one of the function of rotation, and not of individual muscles. The action of the sternomastoid, as a rule, is easily seen; the muscle stands out prominently during the spasm, and becomes very hard to the touch; at times, it undergoes marked hypertrophy. Similar though less marked changes may be observed in the trapezius. The spasm varies in character in different cases. Sometimes it consists of a series of short, jerky movements. At other times it is a long, continuous movement, in which the head and chin are moved through an area of relatively wide extent. In pronounced cases the torsion of the head and neck is so marked as to attain the possible maximum. In exceptional cases the

spasm lasts for many minutes at a time, so that the head is held more or less fixed in one position. Very curiously, in a majority of cases, the muscles of the right side are the ones involved.

Quite commonly the intervals between the attacks of spasm are relatively long, and usually constitute periods of complete or almost complete rest; at other times they are so short that the contractions appear to be almost continuous. The spasm usually ceases during sleep; exceptionally only is sleep interfered with. As a rule the spasm is not accompanied by pain. A dull aching sensation, however, is sometimes referred to the affected muscles, generally the upper part of the trapezius and sternomastoid, and quite frequently to their points of origin on the occiput and mastoid process. The pain is much like a fatigue ache. The patient may have pain, which he refers to the back of the neck and the cervical spine, and sometimes to the dorsal spine; slight sensitiveness to pressure may be met with in these regions.

In rare cases the pain is referred to the upper part of the right arm. Once in a while, points of tenderness may be found over the spinal accessory nerve. Occasionally pressure upon such a point arrests the spasm. At other times pressure-points, not painful in character, such as are met with in facial spasm, are met with in torticollis. They are rare, and when they exist are found usually over the spinal accessory nerve. Very rarely the spasm spreads to the facial muscles, the platysma, the muscles of mastication, and even to the muscles of the arms.

TREATMENT.

The treatment is both medical and surgical. Drugs yield no definite result. Among them may be mentioned antispasmodics such as bromids, belladonna and cannabis indica and tonics such as iron and arsenic. Morphin, as in the case of facial spasm, gives decided relief, but because of the danger of a drug habit, it is not to be used except in extreme cases, and then for short periods of time only; *e.g.*, when the spasm is so severe as to prevent sleep. Fortunately we have another drug, gelsemium, which, while not giving the degree of relief that morphin does, decidedly mitigates the symptoms, and does

this without the formation of a habit. It is a depressing remedy, and should be given with great care. The treatment should be started with 1- or 2- drop (0.06 or 0.12 mil) doses of the fluidextract, given at intervals of four hours. Very gradually the dose can be increased up to 5, 10, or 15 drops (0.30, 0.60, or 0.92 mil). More than 20 drops (1.25 mils) at one dose should not be given. The patient should be closely watched while taking this treatment, and if ocular signs, such as double vision, make their appearance, the treatment should at once be discontinued. By beginning with a small dose, tolerance to the drug is established. The spasm diminishes to a marked extent, and in some cases almost ceases. The relief is so great that it adds much to the comfort of the patient, and the improvement usually continues for some time after the drug is discontinued. It is a good plan whenever possible to give a patient at the same time a course of full rest treatment. The affection occurs most frequently in women who are neurasthenic, and who are therefore greatly benefited in general health by rest treatment; further, if gelsemium be used while the patient is resting, the greatest amount of benefit is secured. Electricity, it should be added, is practically useless.

In very severe cases, surgical interference must be considered. In a large number of instances the spinal accessory nerve has been cut, stretched or resected. Great improvement follows such treatment, but as a rule the spasm returns after a number of weeks or months, though with lessened severity. More extensive operations have been practised; but when we consider that at least ten muscles can be named, in addition to the sternomastoid and trapezius, that are concerned in rotation, we can hardly expect surgery to cure the patient; the best result will probably be a more or less marked improvement.

Now and then the spasm of the muscles of rotation is bilateral. It is seen almost exclusively in children, in whom it is commonly the result of dental irritation. If the muscles of the two sides act simultaneously, there is a movement as of bowing, which is repeated at short intervals. If the muscles of the two sides act alternately, the head is constantly rolled from side to side. Pain does not accompany the affection, but it is very distressing to the family. Occasionally a very bril-

liant result is obtained by lancing the gums. If this measure fails, other peripheral irritation should be looked for, special attention being paid to the intestinal tract.

At times, the spasm which affects the muscles of the neck is tonic in character, the head being held firmly in one position. As a rule, the neck is bent to one side, while the occiput is drawn backward, and the whole head somewhat rotated, the position varying as the action of the trapezius or of the sterno-mastoid predominates. This affection may be the result of irritation of deep structures, such as disease of the cervical spine or meninges. In such cases other symptoms should be present, though frequently the cause cannot be determined. It should not be confused with rheumatism of the muscles producing ordinary wry-neck. The treatment must be based on the cause, when the latter can be discovered. If no cause can be found, counterirritation over the cervical spine may be practised, together with the administration of sedatives. If the case fails to yield, surgical intervention may prove of benefit. Thus, tendons may be cut and the head secured in the normal position by means of a properly constructed apparatus. The treatment of such a case becomes essentially orthopedic. Extension of the cervical spine may also be practised with advantage. Surgical intervention should not be too long delayed, as the tendency is for the head to become permanently fixed in an abnormal position.

Masticatory Spasm. Among the rarer forms of myospasm is masticatory spasm. It is occasionally met with in hysteria. Very rarely it exists as an independent affection. Under such circumstances it is due to some peripheral irritation such as caries of the teeth, neuralgia, or some irritation of one of the sensory branches of the fifth. Very rarely, lesions in distant parts, such as the extremities, are followed by this curious affection. Irritation of the intestinal tract also should not be forgotten. If hysteria has been eliminated, the teeth and the body generally should be carefully explored. If a rheumatic cause be suspected, the salicylates should be administered. If no cause be discovered, electricity, moist heat, counterirritation to the back of the neck or mastoid regions may be employed; and in children a vermifuge may be tried.

Myoclonus. Paramyoclonus Multiplex. This affection was originally described by Friedreich in 1881. It manifests itself by sudden, lightning-like clonic spasms involving the muscles of the trunk and extremities; the facial muscles are rarely involved. The spasms attack the muscles of both sides of the body about equally, though it rarely happens that the corresponding muscles of the two sides contract at the same time. The attacks come on in paroxysms which last for several minutes. The clonus is rhythmic, though its rate varies considerably. No seizures occur during sleep. Voluntary motion may inhibit or lessen them. The tendon reflexes are usually increased. Motor power is intact. Sensibility appears to be normal. The mental condition is normal. The affection usually occurs during adult life. A peculiar form of myoclonus has been described by Unverricht, occurring in families, and bearing a distinct relation to epilepsy.

The important matter in all cases of myoclonus is the differential diagnosis from hysteria. Hysterical myoclonus, of course, offers a good prognosis. True Friedreich's paramyoclonus is a chronic disease, which offers little prospect of cure. No pathologic lesions have been found to account for the affection. Spasmodic phenomena resembling myoclonus, it may be mentioned, have been observed in animals deprived of their parathyroid glands.

In the way of treatment, bromids, arsenic, galvanic and static electricity may be employed. Small doses of thyroid extract are supposed to have been effective occasionally. The general physiologic methods already considered under neurasthenia and hysteria should be thoroughly applied in these cases.

Saltatoric Spasm. In saltatoric spasm, as originally described by Bamberger, the patient passes through a series of springing, jumping, hopping or dancing movements whenever he attempts to walk, the movements usually beginning as soon as the feet are placed upon the ground. The movements are absent in bed. Hysterical stigmata are usually present, and the affection does not constitute a clinical entity, but is only one of the motor manifestations of hysteria. The prognosis is good, and the treatment is that of hysteria.

THE OCCUPATION NEUROSES.

The characteristic symptom of the occupation neuroses is spasm induced by the performance of certain and frequently repeated movements or actions. This form of myospasm is most frequently met with as writers' cramp or scriveners' palsy. To this group are to be added the occupation cramps of telegraphers, pianists, shoemakers, milkers, or those of other occupations which call for the habitual performance of certain movements. The direct cause of the disturbance is overuse and overexertion. However, it is generally evident that the patient attacked is nervously predisposed, is neurasthenic, or is in general ill health. Sometimes more than one individual of the same family suffer from the affection.

In most cases, the patient at first feels only fatigue in the performance of the movement, *e.g.,* in writing. There is a lack of control over the pen; the pen tends to stop or stick to the paper; and the cramp of the muscles finally develops until motion of the pen is impossible. Again, the pen may fall from the hand because of the finger being cramped in extension. However, the flexors of the thumb, forefinger and middle finger are the muscles mainly involved. Later, the wrist and forearm may be affected, and the patient may complain of pain or aching in the hand, sometimes of weakness or tremor. The condition may be so severe that the mere act of grasping a pen may bring on a cramp. The hand and arm are entirely normal for other movements, and it seems probable that the symptoms are directly due to an exhaustion of the neuromuscular mechanism concerned in writing.

TREATMENT.

The treatment should consist of a change in occupation. Frequently this cannot be carried out, and in mild or beginning cases the patient is unwilling or unable to abstain entirely from writing, and in such cases it is imperative to advise the patient as to the manner in which the continuation of writing will prove least harmful. In such cases writing should be performed by full arm and shoulder movements, and not by the fingers and hand. The hand should not rest upon the paper. Again, the handle of the pen may be thrust through a

cork, large and conveniently rounded, and the latter grasped by the palm and the base of the thumb and fingers. Many patients can write more easily with a lead pencil or quill than with a steel pen. In short, anything which changes the detail of the co-ordinations required is helpful.

Treatment should also be directed toward the general health. The relation to neurasthenia and neuropathic conditions is often so obvious that general rest and physiologic methods are indicated, and for these the reader is referred to the section on Partial Rest Treatment. (See p. 583.)

Tonics such as iron, arsenic and quinin are indicated. Local treatment by electricity and massage, while not very effectual, are of some benefit, and encourage the patient by suggestion. That the fatigue is central and not local is shown by the fact that should the patient train his left hand to take the place of the right, the left will also become subject to the cramp. General exercises, not too fatiguing, may be permitted, but these had best be taken along with other physiologic methods indicated under Partial Rest Treatment. The treatment of other occupation neuroses embraces the application of the general principles here indicated.

MYASTHENIA GRAVIS.

Myasthenia gravis is an affection, the characteristic feature of which is a paresis, functional in character, but which closely simulates an organic palsy. It most frequently makes its appearance in a form which closely simulates true bulbar palsy. However, instead of progressing steadily, as does true bulbar palsy, cases of bulbar myasthenia vary from time to time as to the intensity of the symptoms. The latter may disappear altogether for a time, and some cases even recover.

Among the various symptoms we find difficulty of articulation, difficulty of swallowing, ptosis, diplopia, weakness of the muscles of mastication, and weakness of the muscles supplied by the facial nerve. The muscles of the neck, trunk and extremities may be involved. There may even be difficulty in breathing. The patients become fatigued with extreme readiness. There is no muscular wasting, and no reaction of degeneration. However, the muscles soon become exhausted

to faradic stimulation, the responses becoming more and more feeble, until they finally disappear. This is known as the myasthenic reaction. After a period of rest the muscles again respond. There is no anesthesia and no pain. Mental symptoms are likewise absent. Marked improvement may ensue during a remission, and the disease may be months in reaching its greatest intensity.

The diagnosis may be quite difficult in a first attack. However, the course of the symptoms, the occurrence of remissions, the absence of fibrillary tremors, the absence of wasting in the muscles, the absence of electrical changes, the presence of the myasthenic reaction, and the extreme ease with which fatigue can be induced, serve to establish the diagnosis.

The anatomic findings in the nervous system are negative. In the muscles, however, Weigert noted a round cell infiltration, this infiltration having its origin apparently in disease of the thymus gland. These observations have been made by other investigators. Further, Buzzard observed round cell infiltration, not only in the muscles, but also in other organs. He does not, however, regard the disturbance of the muscle function as dependent on this infiltration, nor does he consider the relation of the infiltration to disease of the thymus as established. He regards the muscle symptoms probably as due to some form of intoxication.

TREATMENT.

During an attack the patient should be put to bed, and every precaution taken to avoid muscular exhaustion. The exhaustion, it should be remembered, may involve the muscles of respiration, and lead indirectly to death. As much food as possible should be administered, preferably milk and eggs. Sometimes there is great difficulty in swallowing, so that solid food can be taken only with effort, and at times not without danger to life. In cases in which the difficulty of swallowing is extreme, nasal feeding should be tried.

Massage and electricity, so useful in other asthenic states, are here inapplicable; they still further fatigue and exhaust the muscles. Tonics, especially strychnin, are indicated. The strychnin should be given hypodermically—in small

doses at first, and, if well borne, should be carefully increased. A cautious use may also be made of thyroid extract. Delille and Vincent believe they have obtained good results in one case from the use of pituitary and ovarian extract. Finally, it would seem that if a persistent and overacting thymus is really the cause of the symptoms, the indication is to remove the gland. However, so radical a procedure would here be especially dangerous, as cases of myasthenia gravis bear anesthetics badly.

TETANY.

Tetany is an affection which manifests itself by intermittent tonic spasm affecting symmetrically the muscles of the extremities, particularly those of the hands and forearms, and causing the hands to assume most frequently the position of the "writing" or "obstetric" hand. The disease, as a rule, is ushered in by vague pains, a general sense of uneasiness, and a feeling of weakness and stiffness in the muscles, which is generally most marked in the arms. The prodromal period is short, sometimes only a few hours. Then a tonic spasm of the muscles comes on, beginning in the fingers and spreading up the forearms and arms. The flexor muscles are most affected. The thumbs and fingers become flexed—the proximal phalanges flexed, the middle and distal phalanges extended—so that the hand assumes the writing or obstetric position. This is not, however, invariably the case. In rare instances, the hand may be clenched or the fingers opened out and spread apart; exceptionally, too, the spasm is limited to a single finger. Spasm having been established in the hand, the wrist next becomes flexed, the forearm flexed upon the arm, and the arm adducted to the trunk. The spasm may also involve the lower extremities. The toes become flexed, the foot arched, and the legs extended upon the thighs. The muscles of the trunk and neck may also be invaded. Very rarely the muscles of mastication, of expression, or the muscles of the eyes are involved. The tongue may be affected; and as the patients at times complain of difficulty in swallowing, it would seem that in such cases the constrictors of the pharynx also participate. Spasm of the larynx is a not infrequent and serious accompaniment of tetany. The

involvement of the muscles is almost always symmetrical; it may, however, be more marked on one side, and cases in which it has been limited to one side have been described.

The attack of spasm comes on gradually. It may last for a few minutes or several hours, and, in rare instances, even days. During its height the affected muscles become very firm and hard, and somewhat sensitive to pressure. The spasm may recur on the same day, the next day, or after several days, and during the interval the patient feels quite well, save that he may complain of slight pain and stiffness in the muscles. The degree of spasm varies from one so slight that the patient may be able temporarily to overcome it, to one so severe that the arm, for instance, is firmly held in contracture.

Besides the spasm, which is the most striking symptom of tetany, there are present, as a rule, other important signs. These consist especially of the Trousseau, the Chvostek, and the Erb symptoms. Trousseau's symptom consists of the fact that the spasm may be brought on, if absent, or made more pronounced, if present, by pressure upon or constriction of an affected extremity. If during a passive interval the arm be grasped so that the large nerve trunks and blood-vessels be compressed, the characteristic cramp sooner or later makes its appearance. This symptom when present establishes the diagnosis of tetany. Unfortunately it is absent in about one-fourth to one-third of the cases. Chvostek's symptom consists of an extraordinary susceptibility of the peripheral nerves to mechanical stimuli. Thus, by striking the 7th nerve in front of the ear a light blow, we observe twitching of the facial muscles; most frequently we see movements merely of the angle of the mouth. At times this symptom may also be observed in the nerves of the extremities. Unlike Trousseau's symptom, it is not pathognomonic of tetany. It is occasionally elicited, however, when there are no other evidences of the disease. Erb's symptom consists of a greatly exaggerated electric excitability of the nerves. Very weak currents—*e.g.*, 0.1 to 0.7 milliampère (Pineles)—induce cathodal closure contractions, Ca Cl C; slightly stronger currents, cathodal closure tetanus, Ca Cl Te; An Cl C and An O C follow readily. An Cl Te, Ca O C, and even Ca O Te are observed.

Hoffman's symptom, less important than the others, consists of an increased mechanical and electrical excitability of sensory nerves. Spontaneous pains, however, are also present, variable in degree. They are referred especially to the muscles affected by the contracture. Paresthesia, *e.g.,* formication, slight numbness, is often complained of. Anesthesia is not present. The reflexes are usually normal; they may, however, be plus and rarely minus. In the neighborhood of the joints edema, redness, herpes and urticaria have been noted. Rarely, falling out of the hair and nails, or transverse marking of the nails, accompanies this disease. Muscular atrophy has been noted, though it is unusual. Excessive sweating, duskiness and cyanosis of the extremities, and also puffiness of the face and swelling of the eyelids may be observed. Changes in temperature are rare, but temperatures both above and below normal have been noted. Respiration is unaffected. Very frequently mental symptoms are present; in each case they are those of exhaustion and confusion. An attack of tetany usually lasts several weeks. Rarely, it consists of only a few paroxysms. Again, it may last for months, though more or less prolonged remissions may be present.

The prognosis on the whole is favorable. No fatal case of idiopathic tetany without complications has been observed. When dilatation of the stomach is present, however, the outlook may be very serious. Further, in the tetany that is observed after thyroid and parathyroid extirpation, the outlook is very serious.

It is probable that tetany is the result of some infection. It appears to be favored by bad hygienic surroundings. Though rare in this country, it is quite common in some cities of Europe, *e.g.,* Vienna, and not infrequently occurs in epidemic form. The fact that tetany results from removal of the parathyroid glands suggests an intimate relation between these glands and the disease. However, tetany seems to be of varied origin, and it is exceedingly probable that it may be the indirect result of various toxins; possibly of the action of these toxins on the parathyroid bodies. Thus it occasionally follows infectious fevers, such as measles, scarlet fever and influenza. At other times it is associated with gastro-intestinal disturbances, indigestion, diarrhea, dilatation

of the stomach, or worms in the intestine. Pregnancy and lactation, it should be added, appear greatly to increase the susceptibility to the disease.

TREATMENT.

The patient should have absolute rest in bed. The diet should consist largely of milk and vegetables. Soups, broths and meats should be avoided. Tea and coffee should be excluded. Warm baths may be used to allay the spasm and increase elimination. Extract of thyroid and parathyroid glands has been found of no value. Bromids and chloral in full doses may be given to lessen the spasm. Antipyrin, phenacetin or some of the salicylates may be employed if there be much pain. If the attack is attended by gastric dilatation, the treatment should be directed against the disease of the stomach. Thorough lavage, rectal feeding and other expedients are indicated. Intestinal antisepsis should also as far as practicable be brought about.

The hygienic surroundings of patients with tetany should be carefully considered. The patients should have abundant fresh air, and should not be exposed to cold. In cases with a tendency to recurrence, a change of residence may be beneficial. Mothers who suffer from tetany during lactation should not nurse their children.

PARALYSIS AGITANS.

Paralysis agitans, or Parkinson's disease, is an affection which manifests itself by a tremor, usually passive in character, by a tendency to fixation in posture, by rigidity of muscles, and by a peculiar propulsive gait.

The etiology of the disease is unknown. It usually makes its appearance in the latter half of life; less frequently it is noted in relatively young individuals. Men appear to suffer somewhat more frequently than women. A great variety of causes have been assigned; *e.g.*, grief, worry, mental strain, shock and overexertion.

Nothing is definitely known of the pathology of the disease. The changes found at autopsy are largely those found in old age; such as arteriosclerosis and associated changes in the

nerve centers. The recent findings by Ramsey Hunt of extensive changes in the basal ganglia, apparently degenerative in their nature, are very suggestive as regards the symptoms, but, on the other hand, the positions and rigidity, especially of the hands and arms, remind one of similar phenomena resulting from parathyroid extirpation, and suggest the possibility of parathyroid disease.

The disease comes on gradually. It may be preceded by pains, paresthesia or fatigue sensations. The first decided symptom is tremor. This usually begins in the hand and fingers of one side. Very frequently it begins in the foot. At first it may be slight and not persistent. However, in time it becomes established, and is almost constant. Soon rigidity appears in the muscles, together with weakness and slowing of movements. The tremor having begun in the arm of one side may spread to the leg on the same side; or it may first involve the opposite hand and fingers, so that both arms are affected before the tremor is noted to any degree in the legs. The patient stands or remains seated in a fixed position. The face is wooden or mask-like, the trunk is inclined forward, the arms somewhat adducted, the forearms partially flexed, the hands and wrists moderately flexed or extended, while the fingers assume the position of the writing hand. The motion of the thumb and fingers suggests pill-rolling. While the tremor is most marked in the extremities, it may also involve the head, the jaw, the lips and the tongue.

The tremor is passive, that is, it persists while the part is at rest, and is temporarily arrested by voluntary motion. In all his movements the patient is slow. Again, in walking he very frequently shows a tendency to propulsion; he leans forward, his steps are short, and he may even be forced to run. He may fall unless he is able to catch hold of some support. If in walking he turns around, the head and trunk turn as one piece, as in a statue. As the disease progresses, the rigidity, fixation and muscular weakness become more pronounced until the patient is forced to remain in his chair or bed, physically helpless.

There are no sensory losses. The tendon reflexes are normal or slightly exaggerated. There is no ankle clonus or Babinski sign. Mental disturbances are rare, but mild con-

fusion or depression may make their appearance late in the disease. The patient may be slow in answering questions, or the words may come quickly or in an explosive utterance. The disease is of long duration; fifteen or twenty years are not uncommon. Death results from exhaustion or visceral complications.

TREATMENT.

Paralysis agitans is one of the few diseases in which rest treatment is not applicable. If possible, the patient should lead a quiet life, preferably in the country, free from mental and physical strain. Simple and abundant food and a full amount of sleep are of course indicated. Gentle but systematic exercises should be instituted and persisted in. The patient usually enjoys gentle movement, though from the nature of his affection he tends to remain quiet. The exercise should be active or passive, according to the case, and should embody the principle of precision, as in those devised by Frankel for the treatment of tabetics. The tendency of the patient is to a gradually increasing fixation, and this the exercise seems to delay. The mere fact of being in motion has a good psychic effect on the patient. Thus he feels better when he is seated in a train, or in an automobile. Sometimes a woman patient will take great comfort from the continuous use of a rocking chair. Other therapeutic procedures, such as massage and electricity, are as a rule badly borne. This is also true of hydrotherapy.

Medicines are of little value. The occasional use of the bromids may be permitted to relieve depression temporarily. Hyoscin or scopolamin $\frac{1}{200}$ grain (0.00033 Gm.) three times daily somewhat lessens the tremor, but fails after a while.

Parathyroid extract $\frac{1}{20}$ to $\frac{1}{10}$ grain (0.003 to 0.006 Gm.) three times daily, is also of temporary benefit in diminishing the tremor; it loses its effect in ten days or two weeks. Tonics are occasionally useful. Strychnin, for obvious reasons, should not be given.

FUNCTIONAL TREMORS.

Among functional tremors, we must distinguish the tremors of neurasthenia, of hysteria, hereditary or familial tremors, senile tremor, and, finally, a tremor classifiable under

none of these heads, which is observed from time to time in neuropathic individuals. The tremors of neurasthenia and hysteria have already been considered. Hereditary or familial tremor is a rather rare affection, the cause of which is unknown. Frequently the family history is entirely free of a neuropathic taint. Occasionally, we meet a history of alcoholism, mental disease or epilepsy in the ancestry. It affects both sexes equally and is transmitted by males and females alike. It usually appears in youth; rarely does it begin at a more advanced age. It is characterized by rhythmic movements, of small extent, which vary from about three to nine in a second. In type it is an intention tremor, though in some cases it is present in a less pronounced form during rest. Occasionally it may be suppressed for a time by the will of the patient; it becomes exaggerated during voluntary movements and during effort. It is most frequently seen in the hands, less frequently in the feet, and rarely in the face, the tongue or the head. Sometimes it becomes more marked with advancing age; in other cases time produces little change. Sometimes there are remissions; infrequently it disappears. The disease is transmitted from generation to generation, and is often present in several members of the same generation. It is not deleterious to life, and does not interfere with the patient's occupation except in those cases in which it becomes more marked as time passes. It is made worse by fatigue, and improves with rest.

Senile tremor affects chiefly the head and arms; it is a tremor of small extent, which is increased by voluntary movements, and which almost or entirely disappears during rest. It is not influenced by treatment.

Simple idiopathic or neuropathic tremor is not a frequent affection and appears to be allied to the myokymias and tics. It is found mostly in patients neuropathically predisposed. It generally attacks the hands and head, and less frequently the muscles of the face and tongue. It is a fine tremor, diminishes during rest, and is made worse by voluntary motion or excitement. Sometimes patients attribute the tremor to fright or shock. Occasionally it disappears for a time; more often it is persistent. It rarely causes the patient any annoyance, and does not respond to treatment.

PART II.

ORGANIC DISEASES OF THE NERVOUS SYSTEM.

In the treatment of organic nervous diseases, the general principles considered in the treatment of functional nervous diseases are, of course, also applicable in greater or less degree. Rest, massage, exercise in so far as it may be practicable, bathing and feeding are equally to be borne in mind. That the benefit to be derived from these procedures is much less pronounced goes, of course, without saying, and that they are most productive of results in comparatively mild cases, and more especially in affections of the cord, peripheral nerves and muscles is equally evident. Nevertheless, they should whenever practicable be employed. In the following sections the special points in the treatment of the various organic affections have been briefly stated. Necessarily the therapeutic results are very limited when compared with those achieved in the field of the functional diseases.

DISEASES OF THE BRAIN AND ITS MEMBRANES.

DISEASES OF THE DURA MATER.

The dura mater is composed of 2 layers; an inner thin layer covered with endothelium, and an outer thicker layer, which serves as the periosteum for the bones of the skull. Inflammation of the dura mater is termed pachymeningitis, either external or internal according to the layer involved.

External Cerebral Pachymeningitis. Inflammation of the outer layer of the dura is secondary to some other condition, such as trauma to the head, caries of the cranial bones due to syphilis, middle ear disease, or infections of the scalp, *e.g.*, erysipelas. Sometimes no cause can be found. In acute cases, pus may form between the dura and the skull. In the chronic or fibrous form, adhesion often occurs between the dura and the skull.

The *symptoms* do not definitely point to the disease. They consist of headache, vertigo, delirium, and perhaps convulsions, symptoms that are present in many other affections. The *diagnosis* commonly follows the recognition of the other conditions present.

The *prognosis* in the acute form is grave. In the chronic form, although response to treatment is poor, death is not likely to result.

The *treatment* of the acute form is almost entirely surgical. In the chronic form, especially if due to syphilis, mercury and iodids are indicated. Counterirritation at the back of the neck may be employed. These means failing, surgical treatment in the form of trephining may be resorted to.

Internal cerebral pachymeningitis may be met with in two forms, purulent and hemorrhagic. Commonly the purulent form is merely a complication of a purulent infection of the outer layer or of the pia, and need not further detain us here. The hemorrhagic form is more common; it is found as a complication in paretic dementia, senile dementia, and chronic alcoholism. It may occur in scurvy, pernicious anemia and other diseases of the blood, syphilis (congenital or acquired), tuberculosis, nephritis, and cardiac disease; it is sometimes, although rarely, met with as a complication in the exanthemata. Finally, it may be traumatic. It occurs most frequently in males and in those of advanced years.

Two views as to the origin of pachymeningitis hemorrhagica are held; first, that an inflammatory exudate occurs, with the subsequent rupture of vessels in this exudate; second, that a hemorrhagic exudation takes place, with subsequent organization and repeated hemorrhages. The latter seems the more plausible explanation, especially when it occurs as a mere trophic disturbance, *e.g.*, in paresis. It is commonly associated with atrophy of the convolutions.

The *symptoms* of the hemorrhagic pachymeningitis are usually vague in character, and are met with as complications of other conditions. They are mainly those of cerebral pressure, and are slight or severe, depending upon the extent of the lesion. The patient may have headache, vertigo, loss of memory, and possibly stupor. He may be hemiplegic or hemiparetic. He may have unilateral convulsions.

The *diagnosis* may be impossible because of the underlying disease with which the pachymeningitis is associated, and on account of the predominant symptoms, *e.g.*, if uremia also be present, the clinical picture relates chiefly to this intoxication. Often the diagnosis can only be conjectural. In senile dementia or in chronic alcoholism, it may attain a certain degree of probability.

The *prognosis* is unfavorable, both from the standpoint of the disease itself and from that of the diseases with which it is associated.

The *treatment* is necessarily that of the underlying and associated conditions. If the diagnosis of a recent hemorrhagic exudation is made—which is doubtful—it may, of course, be treated symptomatically; for example, by elevating the head and applying an ice-cap. Lumbar puncture and trephining are expedients of doubtful value.

ACUTE CEREBRAL LEPTOMENINGITIS.

The term signifies an acute inflammation of the pia-arachnoid covering the brain. The causes are to be sought in bacterial infections; *e.g.*, streptococcus, pneumococcus, bacillus tuberculosis, the spirillum of syphilis, the gonococcus, and the diplococcus intracellularis. It may be the sequel of some acute infectious disease, especially of pneumonia, of which it is not an uncommon complication. It may arise from middle ear disease, and affections of the nose, throat, nasal sinuses, and tonsils. A brain abscess also may reach the surface, and thus cause meningitis. It is met with most frequently in the young.

There is an intense cellular infiltration of the pia-arachnoid, the cells being chiefly mononuclear in syphilitic and tubercular forms, and polynuclear in the purulent varieties. If the process lasts a sufficient time, the membranes and blood-vessels are thickened, and small hemorrhages may be found in the membranes. The brain cortex may be softened and contain hemorrhagic foci.

Prodromal *symptoms* of languor, headache, vomiting, and fever may be present. The attack may be ushered in suddenly with a chill, fever, and violent and persistent headache. De-

lirium soon is manifest, and in severe cases is followed by stupor. Retraction of the head, rigidity of the muscles of the back of the neck, retraction of the abdominal muscles, and inability to extend the leg on the thigh when thigh is flexed on the abdomen (Kernig's sign) are prominent symptoms. Palsies of the cranial nerves may be present, and point naturally to a basilar meningitis. Muscular twitchings and convulsions are common, the latter being due to implication of the motor areas. Optic neuritis may occur. The pupils may be contracted or dilated and unequal.

With a clear history the *diagnosis* should not be difficult; however, the toxins of the infectious diseases, especially typhoid fever and pneumonia in a child, may cause cortical irritation closely simulating meningitis. The examination of the spinal fluid is often of value in establishing a diagnosis.

TREATMENT.

The patient should be kept quiet in bed, and the bowels freely opened, preferably with calomel. The ice-bag should be applied to the head. Pain must be controlled by bromids, acetphenetidin, and, if necessary, by opium. Benefit may be derived from lumbar puncture. If the patient stands drainage of the cerebrospinal fluid well, the lumbar puncture may be repeated with benefit every second or third day. If the disease has its origin in a purulent focus in some other part of the body this focus must also be treated.

In the epidemic form, that due to the *Diplococcus intracellularis*, the antiserum developed by Flexner should be used; this should be injected into the subdural space. If the spinal fluid is turbid or purulent it is wise to use the antiserum at once and not to wait for a bacteriologic diagnosis. The antiserum must be kept cold until ready for use, and after it has been warmed to the body temperature, 15 to 45 mils (4 to 12 f3) may be given at one dose, except in young infants when smaller doses should be used. The injections may be repeated daily for several days, according to the condition of the patient and the findings in the spinal fluid. So long as meningococci are found the injections should be continued. In the fulminant and very severe cases the injection may be given twice during the first twenty-four hours.

HYDROCEPHALUS.

Hydrocephalus is characterized by an increase—usually a very great increase—in the amount of fluid in the ventricles. So-called external hydrocephalus occurs as a condition secondary to brain atrophy, *e.g.,* in paresis, and does not demand special consideration. Internal hydrocephalus may be congenital or acquired.

The congenital form commonly is the result of a malformation of the brain, having its origin in embryonic life, and may be complicated with porencephaly. The etiology is not well understood, but occlusion of the aqueduct of Sylvius may play a rôle; at other times it may be due to disease of the ependyma. Syphilis is the underlying cause in many congenital cases, and alcohol also may be the provocative factor. When acquired it may be due to any cause which obstructs the outflow of fluid from the ventricles; for instance, a basal meningitis or a tumor pressing upon the aqueduct. The disease may be present at birth, or may show itself within the first year of life.

The characteristic *symptom* is a gradual increase in the size of the head, which in some cases becomes enormously enlarged. The fontanelles bulge, and the child becomes restless and irritable. The eyes are prominent; nystagmus, strabismus, and optic atrophy may be present. The child may or may not learn to walk and talk. A spastic gait may develop. Convulsions are likely to occur. The outlook is bad, except in mild cases.

Treatment is very unsatisfactory. If due to syphilis results may be obtained from thorough treatment. There is also reason to believe that this treatment will be aided by systematic lumbar puncture and drainage of the cerebrospinal fluid. Puncture of the corpus callosum (*balgenstich*) may be resorted to. The results, however, are not very encouraging. The removal of tumors, *e.g.,* of the pineal gland, compressing the aqueduct is still a problem confronting the surgeons.

BRAIN ABSCESS.

Brain abscess is caused by infection carried to the brain in the blood stream, or by the extension of inflammation from contiguous infected structures. Abscesses occur most fre-

quently in the cerebrum, either in the temporal or frontal lobes; those due to ear disease usually are located in the temporal lobe or in the cerebellum.

The *symptoms* of brain abscess may run either a rapid or a slow course, the former type usually following trauma. If there are no focal symptoms the clinical picture may be mistaken for meningitis. The focal symptoms when present are revealed as a hemiplegia, aphasia, localized convulsions, or by other signs; or if in the cerebellum, by incoördination and other cerebellar symptoms. Optic neuritis may occur. Death may occur in a few days.

The chronic form usually is associated with ear disease. The symptoms may not be present for months at a time. The patient may suffer from headache and vertigo; he may have acute attacks of severe headache, and vomiting or convulsions, and apparently regain his usual health. The temperature is normal or subnormal. The pulse may be slow, although this change is not met with as frequently as in brain tumor. In the terminal stage, the symptoms may assume an acute character or there may be epileptiform seizures, followed by coma and death.

The *diagnosis* often is difficult. Much depends upon the history of the case, and inquiry as to disease of the middle ear or of the frontal or other sinus is essential. The temperature remains normal or subnormal unless there is an associated meningitis. Focal symptoms and the direct evidence of disease of the ear or sinuses are of great value. In the differentiation between an otitic abscess in the temporal lobe and cerebellum the Bárány method may be of value. (See section on Vertigo, p. 615.)

The outlook is discouraging. The *treatment* is necessarily surgical, and should be instituted as soon as the diagnosis can be accurately made. In very rare cases the abscess empties itself spontaneously, as through the nasal passages. One of the great dangers from brain abscess is the development of a secondary purulent meningitis.

THROMBOSIS OF THE CEREBRAL SINUSES.

Marantic thrombosis is caused by general disease resulting in cardiac weakness, changes in the coagulability of the blood, and injury of the inner wall of the sinus.

Inflammatory thrombosis is the result of septic infection, usually from caries of the petrous portion of the temporal bone. This leads to purulent leptomeningitis, extradural abscess, and brain abscess.

Marantic thrombosis usually occurs in the longitudinal and transverse sinuses. The consequences are congestion, edema, and hemorrhage into the brain. The brain being enclosed in a rigid bony cavity, the arterial flow of blood will decrease because of the congestion and edema.

Mental dullness, stupor, and coma will result; and at times delirium, convulsions, and palsies. The diagnosis is difficult, since the symptoms resemble meningitis.

Thrombosis of the transverse sinus may show edema behind the ear. In thrombosis of the longitudinal sinus there may be engorgement of the veins passing from the parietal to the temporal region. The nose may bleed freely as a result of the congestion of the veins of the nose. In thrombosis of the cavernous sinus there is edema of eyelid and face; exophthalmos, engorgement of retinal veins, and edema of the disc may appear. Paralysis of the eye muscles and neuralgia may be present. Inflammatory thrombosis of the cavernous sinus usually is caused by inflammations of the orbit and face. The diagnosis is made more certain by discovery of the primary focus of infection.

The *treatment* is most discouraging. Marantic thrombosis demands rest, and treatment of the anemia or other general conditions. After the thrombus has formed little can be done by medication. Purulent thrombosis demands removal, by operation, of the part of the sinus affected.

CIRCULATORY DISTURBANCES OF THE BRAIN.

CEREBRAL ANEMIA.

For a long time the symptoms of neurasthenia were erroneously ascribed to cerebral anemia. True cerebral anemia oc-

curs merely as a symptom of other conditions. Its diagnosis as an independent affection is practically never made in the modern clinic.

Anemia of the brain occurs as a complication in diseases of the blood—chlorosis, pernicious anemia, and leukemia; in cachectic conditions; in inanition; it may result from the pressure of a brain tumor within an unyielding skull, from the sudden dilatation of the blood-vessels in other portions of the body, as in emotion or shock, and from the lowering of intra-abdominal pressure by the removal of a large tumor or an extensive ascites; from sudden and grave hemorrhage, heart failure, or other causes that greatly diminish the blood supply to the brain.

If of sudden onset, the patient complains of *symptoms* such as darkness before the eyes, vertigo, tinnitus, headache, and muscular twitching; in severe cases there is mental torpor, syncope, and loss of consciousness. The skin becomes pale and cold, and the heart's action rapid. The prognosis is, of course, that of the underlying affection.

The *treatment,* in an acute case, as for instance in an ordinary fainting spell, consists of placing the patient in the horizontal position with the head low. Stimulants, aromatic spirits of ammonia, whisky and hot water, or the administration of strychnin, digitalis, or atropin may be indicated. Hot coffee may be given by the rectum. Stimulation of the surface of the body by vigorous friction, slapping with a wet towel, and sinapisms to the epigastrium may aid in bringing about a reaction. In chronic cases, the treatment is that of the underlying disease.

CEREBRAL HYPEREMIA.

Like cerebral anemia, hyperemia of the brain is met with in practice largely as a complication of other conditions, particularly disease of the heart, and at times of the lungs. Here the congestion is passive in character. It is possible that active hyperemia occurs as a complication of some of the acute mental disturbances, but the latter are essentially toxic in origin. It occurs possibly also as a result of large doses of quinin, and the nitrites also have a direct effect in bringing about dilatation of the vessels of the brain. The amount of

blood present in the cranial cavity is also influenced by severe coughing, such as that excited by attacks of whooping-cough. Brain tumor, sinus thrombosis, and pressure upon the veins of the neck have a similar and more continuous influence.

Hyperemia of the brain is probably accompanied by *symptoms* of headache, tinnitus, vertigo, by a sensation of fullness of the head, and possibly by epileptiform attacks. The symptoms are made worse by lying down; as a whole, however, they form a very subordinate group in comparison with those of the underlying disease.

The *treatment* is that of the underlying disease. If the symptoms are pronounced, however, they may demand special attention. In such case, the head should be elevated, and cold applications used. A hot mustard bath of the feet and hands may be applied. In passive congestion, free venesection may be indicated. Mental effort and excitement, severe coughing, and straining at stool should be avoided. If the blood pressure is high, it should be gradually reduced, by appropriate measures. This is best brought about by a restricted diet, largely of milk, and by the living of a quiet life. The use of the iodids in small doses for a long period may be of benefit. Ergot has been recommended on theoretical grounds, but it is probably of little use.

CEREBRAL APOPLEXY.

The term apoplexy, which literally means a striking down, is applied to the symptoms produced by embolism, thrombosis, or hemorrhage of the brain; at times similar symptoms are noted due to effusion and to toxic states.

Disease of the vessels is the common cause of hemorrhage and of thrombosis. Embolism is usually associated with acute or chronic endocarditis. Among exciting causes are violent mental or physical exercise, over-eating and over-drinking, and other causes which raise the blood-pressure. Transient apoplectiform attacks occur in paresis, uremia, and at times in brain tumor. Changes in the blood that make for excessive hematopexis, together with a feeble heart action, predispose to thrombosis.

In general terms, arteriosclerosis is the most common underlying lesion. Any causes which weaken the vessel wall, such as an endarteritis or periarteritis, may lead to rupture. Not infrequently a periarteritis leads to the formation of small aneurisms, so-called miliary aneurisms, the rupture of which gives rise to hemorrhage. Brain hemorrhage most frequently takes place in the region of the internal capsule. After a hemorrhage, a clot forms, softens, and absorption takes place. Inflammation in the tissues about the clot occurs, and may lead to the formation of a cyst, or instead proliferation of connective tissue with the formation of a pigmented scar may result. The lesion tears in greater or less degree the fibers of the motor pathway, and the fibers below the lesion degenerate. If an artery is obstructed by a thrombus or an embolus, degeneration and softening of the area supplied take place, as there is little or no collateral circulation in the arteries of the brain. If the area be small it may gradually become absorbed; if large, inflammation of the surrounding tissue may lead to the formation of a retaining wall and a cyst. If an embolus arises from some infected source, such as a septic endocarditis, a brain abscess is to be anticipated.

The *symptoms* of hemorrhage, embolism, and thrombosis so closely resemble each other as to render a differential diagnosis very difficult. Cerebral embolism comes on very suddenly and the period of unconsciousness usually is short; with the attack also we observe the associated heart lesion. Cerebral thrombosis usually comes on slowly with prodromal symptoms, such as vertigo, paresthesias, and headache; it may, however, develop rapidly. Unconsciousness may or may not be present. The onset of a cerebral hemorrhage usually is sudden with profound unconsciousness; the symptoms may however, supervene very gradually—so-called ingravescent hemorrhage.

In all of these conditions the lesion usually is on one side of the brain; the motor areas of the affected side are interfered with, and a paralysis of motion occurs upon the opposite side of the body. During the acute stage of the attack the pulse is usually slow and full, the blood-pressure increased, and the breathing stertorous.

The head and eyes are commonly turned away from the paralyzed side (conjugate deviation). The pupils are irresponsive to light, and may be contracted. The muscles of the limbs are relaxed, those on the paralyzed side more than those on the sound side. The temperature at first may be subnormal, but later it tends to rise 1 or 2 degrees above normal. Frequently the axillary temperature of the paralyzed side is a degree higher than that of the sound side.

If the patient's condition grows worse, the pulse, at first slow and full, now becomes rapid, the unconsciousness deepens, the temperature rises rapidly, and the respiration assumes the Cheyne-Stokes type. In cases with a favorable outcome the patient becomes conscious after a few hours or possibly a day, and then one side of the body, including the lower half of the face, is noted to be frankly paralyzed. In right-handed individuals, if the lesion be on the left side, aphasia is present.

The tendon reflexes of the paralyzed side are diminished or absent at first; though a Babinski sign may be noted very early. Later, *i.e.*, within about a fortnight, the tendon reflexes become exaggerated, and the Babinski sign is commonly pronounced.

TREATMENT.

As a preventive, persons with atheromatous arteries, who have passed middle life, should avoid overeating, overdrinking, and strenuous physical exercise. The prolonged administration of small doses of the iodids seems to have a beneficial effect upon the arteriosclerosis. The persistent administration of small doses of thyroid extract is similarly of value.

In the treatment of the attack, after an apoplexy has occurred, the patient should be kept quiet in bed, with the head slightly elevated. He should be turned on his side part of the time, so that the paralyzed tongue may fall forward. If the blood-pressure is high, venesection may be of benefit. Ordinarily full doses of aconite, or, better, veratrum viride, will quiet the pulse. An ice-cap may be applied to the head, and heat, in the form of hot bottles, or hot mustard cloths, to the feet. If the bladder is distended, the patient should be catheterized. A brisk purgative, such as Epsom salts, should be

administered if the patient is able to swallow; if not, the bowels should be moved by enemas.

In fatal cases the coma deepens, the breathing becomes Cheyne-Stokes in type, and the temperature rises. In favorable cases the coma gradually becomes less profound, and in due course of time consciousness returns. The paralysis of the affected side is now more plainly evident. If anesthesia be present, it indicates implication of the posterior third of the posterior limb of the internal capsule. The speech may be thick and indistinct; if the paralysis is on the right side, aphasia is very likely to be present. After three or four weeks, some power returns in arm and leg. The leg usually improves more rapidly than the arm, and the proximal muscles more than the distal. The reflexes now become exaggerated and the palsied muscles are spastic. In this stage but slight further improvement takes place, and little can be accomplished by treatment. The iodids may be given in moderate doses. Electricity is of no value. Massage, and especially passive movements of the paralyzed limbs, may aid in preventing severe contractures.

Apoplexy is usually followed by some degree of mental impairment—sometimes very slight, sometimes pronounced—together with associated emotional disturbances and irritability. Nerve sedatives, such as the bromids, used occasionally, may be of benefit.

BRAIN TUMOR.

Brain tumor occurs in persons of all ages, and males appear to be more subject to such neoplasms than females. The causes are as obscure as that of a tumor in other parts of the body, and any variety may occur in the brain. The most common are tuberculous tumors, sarcoma, glioma, gliosarcoma, cyst, carcinoma, and gumma. In a brain in which a tumor has developed, the membranes are likely to be tense and the convolutions flattened from pressure. The convolutions in the region of the growth do not pulsate. The ventricles are prone to become distended with fluid, and the brain-tissue wet and heavier than normal. A zone of softening frequently is found about the tumor.

The *symptoms* of brain tumor may be grouped as general and focal. The general symptoms consist of headache, vomiting without gastric disturbance—so-called projectile vomiting —and optic neuritis followed by optic atrophy. Vertigo is not infrequent. Mental changes, either slight, or marked, may also be present.

The focal symptoms depend upon the part of the brain in which the growth occurs. Thus, in tumors of the motor area, focal or localized epileptiform convulsions may occur; or there may be various forms of aphasia due to left-sided lesions, or characteristic disturbances of the visual fields due to invasion of the optic pathways, or of the occipital lobe. In estimating the value of localizing signs, it should be borne in mind that late in the history of a patient, focal symptoms due to increased intracranial pressure and circulatory disturbances, may point to parts of the brain not attacked by the tumor.

The *diagnosis* of brain tumor depends, of course, on the presence of the symptoms just considered, and in detail upon a correct application of the facts of cerebral localization.

TREATMENT.

If the tumor produces clearly defined focal symptoms, and if it is so situated that it can be removed without too great a danger from hemorrhage, and from trauma to the brain, the surgical treatment of the condition is, other things being equal, satisfactory. If the tumor should be a gumma, which fact may be strongly suggested by the history of the case, and by the blood and spinal fluid findings, the use of full doses of mercurials and the iodids may be followed by good results. A course of intravenous injections of salvarsan should also be instituted. Unfortunately, the larger number of brain tumors are not specific in origin. However, most cases are benefited by the iodids, given first in small doses, and gradually increased to the point of tolerance. The treatment is, however, only palliative, and the benefit temporary, for as the tumor becomes larger, the symptoms again become more pronounced.

A brain tumor may grow slowly, and localizing symptoms may be absent, or at most illy defined, and the symptoms in such cases must be treated as they arise. Optic neuritis should be treated by the relief of intracranial pressure. Lum-

bar puncture guardedly performed may be of value, from 4 to 6 mils (1 to 1.6 f3) of spinal fluid being withdrawn at intervals of every day, or every other day, and if no unfavorable symptoms make their appearance, the amount withdrawn should be progressively increased. Marked subsidence of the swelling of the optic disc may follow this expedient. Sudden and large withdrawal of spinal fluid may be followed by a fatal result. The operation of cerebral decompression is more radical, and gives prompt relief to both the headache, and swelling of the optic nerve. Right subtemporal decompression is the operation most frequently performed. Subtentorial decompression is adopted, when the symptoms suggest the possibility of the tumor of the posterior cranial fossa. Unfortunately, many cases come to the hospital, or to the hands of the neurologist so late that decompression fails to preserve the vision of the patient. When optic atrophy has been established, decompression has little to offer save the relief of pain. However, in cases in which the tumor can be localized and the skull opened over the tumor, even though the latter proves to be inoperable, the intracranial pressure and headache are relieved for a time.

Headache, vomiting, and vertigo may be so severe as to demand symptomatic treatment, and here the bromids, the coal-tar products, and even morphin, and scopolamin may be employed. Severe and frequent convulsions may be controlled by the bromids and chloral.

DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

Inflammatory diseases of the spinal meninges usually are associated with similar disease processes in the meninges of the brain. An exception to this is hypertrophic cervical pachymeningitis, which occurs as a distinct affection.

Hypertrophic Cervical Pachymeningitis. Trauma, tuberculosis, and especially syphilis, appear to be prominent factors of this affection, while other potential causes, such as rheumatism and alcohol, deserve less credit.

The early *symptoms* consist of pains in the back of the neck, sometimes in the occipital region, frequently between

the shoulders; a feeling of tension, and at times of stiffness; sensitiveness of the cervical spinous processes to percussion; and various pains, and paresthesias, due to implication of the roots. Inasmuch as the lower cervical region commonly is most affected, pains are quite frequent in the course of the median and ulnar nerves. Wasting of the muscles supplied by these nerves, the flexor group of the forearm, and the small muscles of the hand, subsequently develops, and, finally, symptoms of compression of the cervical cord appear. Spastic paralysis of the legs, anesthesia, and sphincter disturbances may be noted. The course of the disease is slowly progressive, the palsies, muscular atrophies, and sensory losses gradually increasing. Sometimes arrest of progress is observed.

A marked hypertrophy, or thickening of the dura mater is present, and as this hypertrophy takes place, the growth implicates, on the one hand the periosteum, and, on the other, the pia-arachnoid, the nerve roots, and the cord; and the newly formed tissue binds membranes, roots, and cord into a single mass.

If the disease is due to syphilis, vigorous antiluetic *treatment* should be instituted. Otherwise the treatment must be symptomatic. In rare cases, surgery may be justified, free laminectomy being the operation of choice. If indicated, it should be done early, and before the cord and nerve-roots have been too greatly damaged.

Spinal leptomeningitis usually is part and parcel of a general cerebrospinal meningitis. In rare cases it occurs as a local process within the spinal canal, due to malignant, tubercular, syphilitic, or other infection. The epidemic variety may be limited to, or most pronounced in, the spinal meninges. It may occur as a sequel to, or a complication of, the infectious diseases, and in this event usually is associated with myelitis.

Dilatation of the blood-vessels of the meninges appears to be present in the beginning of the disease, and is followed by a serous, or seropurulent exudate. The membranes become the seat of inflammatory infiltration and deposit, and the spinal fluid becomes turbid from the presence of formed elements. Whenever practicable, it should be submitted to microscopic and bacteriologic examination.

The onset of the symptoms may be marked by a chill and fever, pain and rigidity of the muscles of the back and neck, shooting pains, and general hyperesthesia, retraction of the abdomen, spasms of the muscles of the arms and legs, exaggerated tendon reflexes, and Kernig's sign, *i.e.,* if the thigh be flexed at right angles to the trunk, the lower leg cannot be extended owing to spasm of the flexors. Later there may be palsies, loss of control of the bladder, and various sensory losses.

The *prognosis* is always bad in tubercular cases; those following acute infectious diseases at times terminate in recovery, although permanent damage to nerve-roots or cord may result.

The *treatment* is that of the underlying general infection, such as syphilis, or tuberculosis, and the disease in question should be managed from its particular standpoint. Rest in bed with an ice-bag to the spine is indicated. Counterirritation over the spine may be used later. Lumbar puncture with the withdrawal of cerebrospinal fluid is reported of benefit in the treatment of various forms of meningeal infection. Spinal drainage, of great importance in diagnosis, relieves intraspinal pressure for a time, and is, as a rule, a safe procedure.

In the treatment of the epidemic form, *i.e.,* of cerebrospinal meningitis, a great advance has been made in the introduction of Flexner's serum. (See Cerebral Leptomeningitis, p. 654.)

DISEASES OF THE SPINAL CORD

Diseases of the spinal cord are conveniently divided into two classes; those affecting chiefly certain tracts or systems of neurons in the cord (System Diseases), and those characterized by diffuse lesions throughout the cord (Diffuse Diseases). In the first group the nerve cells and tracts are primarily affected; and in the second group the lesion usually is one primarily of the blood-vessels, and connective tissue elements. This division is open to some objection, as in given instances, *e.g.,* tabes, the lesion is not confined to the nerve elements alone, but also affects the membranes and the blood-vessels.

The most important system cord disease affecting the sensory tract is tabes dorsalis, the treatment of which will be

considered in the section on the treatment of syphilis of the nervous system. (See p. 689.)

A consideration of the motor tract is essential to an understanding of the various motor system diseases of the cord. The upper motor segment or neuron includes that part which begins on the cells of the motor cortex. The individual axons pass, as axis cylinders, downward through the centrum ovale, internal capsule, crus, pons, and cord, and end—through an intermediate short neuron—near the large multipolar cells in the anterior horn of the gray matter of the cord. They are in physiologic continuity with these multipolar cells. The lower motor segment or neuron begins in the multipolar cells in the anterior horns, and continues through the anterior roots as axis cylinders through the nerve trunks to the muscles. The upper neuron may be diseased at any point in its course.

The *symptoms* consist of paralysis and spasticity of muscles, exaggerated tendon reflexes, and the Babinski sign. The latter consists, the reader will remember, of an extension of the toes, especially the great toe, upon irritation of the sole of the foot. There is no muscular wasting, and no change in the reaction of the muscle to the electric current. If the lower motor neuron be affected, the symptoms consist of paralysis, flaccidity, wasting of muscles, diminished or lost reflexes, and a change in the reaction of the muscle to the galvanic current, the so-called reaction of degeneration. In this the normal sequence of response is changed to the opening and closing of the poles, so that in the fully developed case the anodal closure contraction is finally greater than the cathodal closure contraction. Various intermediate stages are, of course, to be noted. Lesions of both the upper and the lower systems present a combination of muscular rigidity with muscular atrophy.

DISEASES AFFECTING THE UPPER MOTOR NEURON.

HEREDITARY SPASTIC PARAPLEGIA.

This is a rare affection, described by Strümpell, which appears in a number of members of a family in the same or different generations. Syphilis appears to play a part in the

etiology in at least some of the cases. The clinical picture may assume either a spinal or cerebral type. The spinal type appears to depend on degeneration, apparently abiogenetic in character, of the motor tracts in the cord.

The *symptoms* are those of lesion of the upper motor neuron, without cerebral manifestations, such as mental arrest and epilepsy. The diagnosis depends on the occurrence of the disease in other members of the family, its gradual development, and the absence of initial convulsions and other cerebral symptoms.

The prognosis is, of course, unfavorable as to cure.

The *treatment* is purely symptomatic. If syphilis is shown to be the cause, some result may possibly be obtained by the specific treatment of this infection.

SPASTIC PARALYSIS OF INFANTS AND CHILDREN.

This condition may present itself as a hemiplegia, a diplegia, or a paraplegia.

The condition is due to disease or injury affecting the motor areas of the brain. The cause of the defect may be traced to prenatal or intrauterine conditions, to lesions incident to birth, and to those occurring after birth. Hereditary syphilis, alcoholism, uterine trauma, and, possibly, psychic disturbance of the mother, may produce a cerebral defect in the child. Premature birth, with an associated lack of development of the pyramidal tracts, and prolonged, difficult births are of etiologic importance. As regards the latter, it should be borne in mind that the use of forceps correctly applied is of less danger than the severe and persistent pressure such as takes place, for instance, when the head ceases to recede between the pains of a difficult and prolonged labor. Cerebral palsy may also follow the infectious fevers. Hemorrhage, thrombosis, embolism, or localized encephalitis also may be the causal factors. Trauma to the head is a rare cause. Hemiplegia usually is not congenital. Diplegia or paraplegia, often spoken of as Little's disease, are, however, commonly congenital. Many of these palsies are apparently abiogenetic in origin, the motor area failing of full development.

The *symptoms* are those of hemiplegia or double hemiplegia, with spasticity and contractures. In diplegia, if the patient be able to walk, the gait is frequently that of a "cross-legged progression."

Inasmuch as most of these patients come under observation after the spastic condition of the muscles has developed, the *treatment* usually resolves itself into methods of ameliorating the spasticity and the deformities caused thereby. The patient should not be allowed to walk early, in order that defective nerve tracts may be spared. Warm baths, gentle massage, and proper exercise counteract the tendency to contracture. Drugs have no marked influence upon the disease, except, possibly, in those cases due to syphilis.

In cases presenting marked contracture, benefit may be obtained by tenotomy. The dorsal nerve roots also may be cut to overcome great spasticity, as advocated by Foerster, and also by Spiller and Frazier. The cerebral hemorrhage occurring in the newly born, and so often causal to these conditions, frequently may be relieved by operation, as done by Cushing. The epilepsy which so frequently accompanies these gross cerebral defects is to be treated on the same general principles as idiopathic epilepsy. The mental condition may be improved by special methods of training, as carried out in institutions for feeble-minded children.

PRIMARY LATERAL SCLEROSIS.

This condition is one of the infrequent forms of organic nervous disease. It has rarely been confirmed by autopsy, although a very few cases have been reported in which no lesion was found except degeneration of the lateral tracts.

The etiology is obscure. Some cases seem to follow syphilis. In some there is a history of general debility, infectious diseases, traumatism, and exposure to cold and wet. Most cases arise between 20 and 40 years of age.

The *symptoms*, relating to a lesion of the upper motor neuron, develop gradually, and may exist for a number of years before the weakness becomes extreme. In advanced cases the patient may, as in infantile diplegia, walk with one leg crossed over the other.

The *diagnosis* rests upon the history and symptomatology. There are no sensory changes, no trophic changes, no implication of the sphincters, and no ataxia.

The *treatment* is unsatisfactory; drugs such as mercury and iodids, silver and arsenic may be tried, but usually fail of effect. Strychnin should not be given. Rest, warm baths and massage may be of service to relieve the spasticity.

DISEASES OF THE LOWER MOTOR NEURON.

ACUTE ANTERIOR POLIOMYELITIS.

This affection, strictly speaking, is not a neuron disease, but it is classed as such, since its most striking symptoms are shown by the lesions of the lower motor neuron. Acute anterior poliomyelitis is an acute infection, affecting the cerebrospinal axis as a whole. (*Cf.* p. 111.)

The exact cause of the disease has not yet been conclusively shown. The virus is known to filter through porcelain and asbestos filters, and to be highly resistant to various destructive agencies. Apparently it is not affected by gastric and intestinal juices, and can resist for three days without injury, a 0.5 per cent. solution of carbolic acid. It is readily destroyed by heat at 50° C. (122° F.) for one-half hour. It appears that the virus gains entrance to the respiratory tract, infects the lymphatics of the upper air passages, and subsequently invades the meninges and the substance of the brain and cord.

In the early stage there is a hyperemia of the cord and meninges, and the blood-vessels of both brain and cord are dilated. There is edema of the cerebrospinal axis, with but little if any increase of the cerebrospinal fluid, which at first, as a rule, is clear, but contains a large number of polynuclear cells. In a few days the mononuclear cells, lymphocytes, predominate. The perivascular lymph spaces of the blood-vessels of the meninges are filled with an exudate of small mononuclear cells—an acute interstitial meningitis. Since the blood-supply of the cord is derived from the vessels of the meninges, the perivascular infiltration extends into the substance of the cord as the disease process advances. This

round-celled infiltration becomes so dense in areas as to obstruct the blood-supply of the nerve cells. Minute hemorrhages also may be present in both the gray and the white matter of the cord. The lack of nutrition of the nerve cells soon results in their atrophy and death. It is possible, of course, that the toxin of the infection may have an especially destructive effect upon the nerve cells.

The *symptoms* at the onset of the disease vary in different patients. A child in previous good health may be put to bed, apparently well, to awake in the morning paralyzed in one or more extremities, while another child, suffering from some acute disease, acquires the anterior poliomyelitis as a complication. Still another may be ill for a number of days before any signs of palsy are seen, and it is thought that, during epidemics especially, numbers of children are attacked and recover without reaching the stage of paralysis. The disease comes on with fever varying from 100° to 103° F. (37.7° to 39.4° C.), often accompanied by vomiting, and at times by diarrhea.

The child complains of headache, or if too young to state his complaint, will show signs of hebetude and irritability. Nasopharyngeal symptoms, such as sneezing and free discharge of mucus, may be present. Because of irritation of the spinal roots, the pain is referred to the joints or extremities; this pain frequently precedes the onset of paralysis. The paralysis comes on quickly, and, as a rule, soon reaches its maximum. It may present itself as a monoplegia, hemiplegia, paraplegia, or all four extremities may be involved. Most frequently it is one leg which is especially affected; less frequently both. All the symptoms of a lower motor neuron lesion are present; rapidly wasting muscles showing the reaction of degeneration, minus or absent reflexes, flaccid paralysis, and cold, livid, clammy skin. Cranial nerve palsies are present in cases in which the disease implicates the medulla.

The *prognosis* as to life is good in the average case; however, cases arising during epidemics, at which times the virulence of the infection appears to be greatly increased, and the meninges and brain severely damaged, give a relatively unfavorable prognosis as to life. Notwithstanding this, a spontaneous and almost complete recovery may occur.

The average case of palsy improves rapidly for the first few weeks, and some children recover almost entirely during this period. Subsequently, however, further progress is slow, but the improvement continues for several years. The prognosis as to recovery from the palsy is not good; even in the most favorable cases some traces of the disease generally will be found to persist as a relic of the attack.

TREATMENT.

Most observers agree that the disease is contagious. In any event a strict quarantine should be maintained for a number of weeks. The premises must be made sanitary. As the exact way in which the virus is transmitted is not known, all possible carriers should be borne in mind. Insects of all kinds, especially flies, should be screened from houses, and food, such as water, milk, and uncooked vegetables, be kept, if possible, free from contamination. That dirty and dusty streets and alleys should receive attention need hardly be mentioned.

As a prophylactic measure, care should be taken to see that the child's mucus membranes, both of the nasopharyngeal and gastrointestinal tracts, are in the best possible condition.

During the acute febrile stage, the child should rest in bed, and, because of the pain, be handled as little as possible. If there is much fever, the ice-cap may be applied. Bromids may be sufficient to quiet the patient, although occasionally it may be necessary to give the coal-tar products, or even opiates. The diet should be such as will easily digest, and the bowels should be kept free. A number of acute cases have been treated with immune serum, that is, with the serum of the blood of children who have recovered from the disease. Favorable results are thought to have been obtained by this method, although a sufficiently large number of cases have not yet been treated to establish with certainty its advantages.

For the paralysis the patient should have absolute rest for three months. The paralyzed limbs are best put up in plaster-of-Paris splints. If it be the leg that is palsied, the limb should be flexed slightly at the knee and the foot fixed at a right angle. If the arm be affected, it should be slightly flexed at the elbow, the hand hyperextended, and the fingers and

thumb put up in full extension. If the shoulder muscles are paralyzed, the arm should be elevated and abducted at a right angle to the body. If the muscles of the back are affected, the patient should have a plaster dressing molded to the back, and in this the subject should lie after it has been padded with cotton. In other words, complete rest promotes absorption and diminishes pressure. The principle is to relieve the weak and atrophied muscles of all strain. The nerve cells also have a better opportunity to recover, if recovery be possible. Light massage is beneficial after the acute stage has passed. Muscle training is of value, but must not be overdone with weak muscles. Braces should be applied to counteract a stronger group of opposing muscles; they should not, however, interfere with muscular development. If the patient can walk without the development of a deformity, no brace is needed. If there be a palsy of the muscles of the back, limited to one side, a deformity of the spine must be carefully guarded against, usually by the application of a corset with steel supporting bars.

Exercise in a bathtub filled with water may be of use, for by this expedient the partly paralyzed limbs can be moved about in the water more readily. Heat and vibration over the spine, applied late, may have some stimulating effect upon the diseased cord. Electricity has little value except to exercise the muscles. Its use in this way, however, should not be overdone.

The treatment should be continued over a very long time. Not infrequently patients who are carried into the clinic with both legs paralyzed may, after treatment for a year or more, be able to walk with the aid of braces.

ACUTE BULBAR PARALYSIS.

An acute bulbar palsy may be due to an infection, in which case it is analogous to acute anterior poliomyelitis; indeed, it is not improbable that in given instances the virus is the same. If the anterior group of motor nuclei are affected, more especially the oculomotor group, the disease is known as polioencephalitis superior. If the nuclei from the seventh to the twelfth cranial nerves are the seat of the lesion, it is spoken

of as polioencephalitis inferior. The symptoms may also be due to hemorrhage or to thrombosis of the vertebral arteries, of the basilar, or of their branches. Embolism and hemorrhage are uncommon causes.

The *symptoms* may come on rapidly. The muscles of the lips, tongue, pharynx, palate, and larynx become paralyzed. Difficulty in swallowing and speaking is present, and the patient may die from the effects of cardiac and respiratory complications. If the condition be that of a superior polioencephalitis, the prognosis as regards life is better. If the symptoms are due to a vascular lesion, they arise with apoplectiform suddenness, and are likely to be fatal.

PROGRESSIVE MUSCULAR ATROPHY.

Progressive muscular atrophy of spinal origin is characterized by a slow, progressive wasting, usually beginning in the small muscles of the hand, or, it may be, in the muscles of the shoulder girdle.

The disease usually occurs during adult life, and no specific cause is known. Heredity is not a factor. More males are attacked than females. Overwork, exposure, spinal concussion, syphilis, and lead poisoning are thought to be factors in various cases. Not infrequently cases are met with giving a positive Wassermann, increased lymphocytes, and globulin in the spinal fluid.

The anterior horn cells atrophy and disappear, with a consequent atrophy of the muscles supplied by these cells. The skin is not affected.

The *symptoms* of the disease develop insidiously. Atrophy may exist in the muscle for some time before the patient notices weakness. It usually begins in the small muscles of the hand, and in years of time extends to the muscles of the arms, shoulders, neck, and trunk. The patient may complain of some aching and paresthesia of the hands. In a small number of cases the atrophy begins in the lower extremities. The reflexes, normal elsewhere, are diminished in the paralyzed muscles. Fibrillary tremors are present in the atrophied muscles, and there is a quantitative change in their reaction to electricity. In advanced cases complete RD may develop.

The *diagnosis* rests upon the history and local signs of atrophy, together with the absence of signs pointing to implication of other parts of the spinal cord or of the nerve trunks.

Chronic anterior poliomyelitis greatly resembles this disease, and from the standpoint of prognosis and treatment may be considered with it. Chronic anterior poliomyelitis comes on more quickly than progressive muscular atrophy, as a rule affects a large group of muscles early, and the paralysis is noted before the atrophy develops.

If syphilis is shown to be the cause, intraspinal specific methods of *treatment* are indicated. (See p. 689.) In other cases general measures must be employed. Rest, moderate exercise, and full feeding are indicated. Properly applied electricity and massage are useful. Full doses of strychnin are believed to be of benefit in retarding the development of the disease.

CHRONIC PROGRESSIVE BULBAR PALSY.

This disease appears to be analogous to progressive spinal muscular atrophy. The degenerative and atrophic process attacks the nerve nuclei in the medulla.

The disease comes on in adult life, and is known as glossolabiolaryngeal paralysis. The muscles of the lips, tongue, palate, pharynx, and larynx gradually atrophy, and difficulty in phonation and deglutition becomes pronounced. The outlook is unfavorable, and death ensues in a few days.

The *treatment* is purely symptomatic, and the measures adopted in progressive muscular atrophy of spinal origin are indicated in this affection. Care in giving food and drink to the patient is necessary to avoid an insufflation pneumonia.

Pseudobulbar paralysis presents symptoms resembling chronic bulbar paralysis, with the exception that muscular atrophy is not present. It is due to bilateral lesions above the lower motor neuron, *e.g.*, in the lenticular nuclei or the internal capsules. In this lesion the outlook is unfavorable, and the treatment purely symptomatic.

AMYOTROPHIC LATERAL SCLEROSIS.

This affection is the most typical example of lesion of both upper and lower motor neurons. The disease is more common in males, and between the ages of 30 and 50. Heredity plays no part. Poisons, such as lead, and infections, such as syphilis, are possible etiologic factors, but no definite cause is known.

There is a slow degeneration of the anterior horn cells, usually beginning in the cervical region, as in chronic progressive muscular atrophy of spinal origin. Associated with this is a degeneration of the pyramidal tracts. In other words, the morbid anatomy of this disease presents a combination of the lesions of progressive muscular atrophy with primary lateral sclerosis.

The *symptoms* are a combination of the symptoms of disease of both the upper and the lower motor neuron—wasting of the small muscles of the hands and arms, with spasticity of muscles, and increased reflexes in the lower extremities. The course of the disease is slow, but progressive.

No *treatment* is known to check the disease. The little that may be done in the way of treatment has been outlined in the sections on lateral sclerosis and progressive muscular atrophy. (See pp. 669 and 674.)

COMBINED SYSTEM DISEASES

The essential feature of this type of disease is a combination of symptoms due to disease of both the posterior and the lateral columns. The anterior horns are not implicated.

ATAXIC PARAPLEGIA.

The terms combined sclerosis and posterolateral sclerosis are used as synonyms for this disease, for which no specific cause is known. It is a disease of adult life, and more common in males than in females. Syphilis may be a cause, and some cases develop during the course of pernicious anemia.

Both posterior and lateral columns of the cord become sclerotic, and the process is often diffused into the mixed zones

of the lateral columns. The direct anterior tracts also are attacked in nearly all cases.

The *symptoms* of the disease develop slowly, as a rule. Ataxia, combined with weakness and stiffness of muscles, is noted, but sensation usually is not impaired. Lightning pains are absent, but dull pain in sacral region may be complained of. Visceral crises are unknown. The knee-jerks are greatly increased; ankle clonus and the Babinski sign are present. Sexual power is lost early, and the sphincters are often impaired. Eye symptoms are not present.

The disease lasts for many years, and recovery never ensues.

If the spinal fluid presents the evidence of syphilis, intraspinal methods of *treatment* should be employed (*q.v.*, p. 689). Otherwise, good hygienic surroundings, warm climate, and warm baths are indicated for the spasticity. Electricity and strychnin should not be used. Arsenic may be given. Courses of the bromids may be useful at times.

HEREDITARY ATAXIA.

This affection may be classed under two types: Friedreich's disease and hereditary cerebellar ataxia.

The lesions in Friedreich's disease are of the combined spinal-cord type, the changes in the cord consisting mainly of a sclerosis of the posterior and lateral columns. Atrophy of the cerebellum has been found in some cases of cerebellar ataxia. Friedreich's disease develops at or before puberty, and is a family disease. The cerebellar form is prone to develop at puberty or after.

Both forms of the disease present *symptoms* of marked ataxia, tremor of the head and extremities, awkward choreiform movements, disordered speech, and nystagmus.

Friedreich's disease presents absent knee-jerks, no optic atrophy, and no palsies of ocular muscles. Cerebellar ataxia is characterized by normal or increased knee-jerks and ocular symptoms.

The *treatment* is merely palliative. The disease is incurable, and means to keep the patient in as good general health as possible are indicated. The contractures which late in the

disease lead to club-foot or other deformities may require surgical interference.

DIFFUSE DISEASES OF THE SPINAL CORD.

The diffuse diseases of the cord have no predilection for either white or gray matter, or for individual neuron systems. The destructive agent comes from without, often by way of the blood-vessels rather than by disease of the nerve tissue itself.

SPINAL MENINGEAL HEMORRHAGE.

In this accident, also termed hematorrhachis, the hemorrhages may be inside or outside of the dura, the usual cause being an injury, with or without fracture of the spine. Severe convulsions, muscular overexertion, or the bursting of a vertebral aneurism are possible causes. The factors of hemorrhage into the cord (hematomyelia) are the same as those responsible for meningeal hemorrhage.

Small hemorrhage into the membranes may cause no *symptoms*. When the hemorrhage is large enough to cause pressure, the symptoms of root irritation will be present, namely, pain, numbness, tingling, and muscular twitchings. The pain may be a girdle pain about the trunk, or may be referred to the extremities. If the pressure increases, palsy and loss of sensation will supervene. Disturbances of the sphincters may occur. The symptoms rapidly reach their height, and then subside as the blood is absorbed. Lumbar puncture will reveal blood in the spinal fluid. Recovery may take place to a great extent, or the patient may be left with damage to his spinal cord, the result of extensive hemorrhage or of severe pressure.

The symptoms of hemorrhage into the cord substance greatly resemble those of hematorrhachis. They are of sudden onset, with numbness, tingling, and a quickly oncoming paralysis, both of motion and sensation, below the seat of the lesion. Hemorrhage into the cord is prone to attack the gray matter, and to extend for long distances up and down the cord rather than transversely. Lumbar puncture does not reveal blood in the spinal fluid. As the clot is absorbed, a cavity in

the central part of the cord may be left, resembling syringomyelia.

The history of trauma, a clean record as to syphilis, and the absence of fever, together with the above symptoms, will suggest the diagnosis. As to *treatment,* if lumbar puncture reveals blood in the spinal fluid, and the *x*-ray picture shows a fractured vertebra, surgical interference, other things being equal, is indicated. If surgery is to offer any hope of relief to a badly compressed spinal cord, the operation should be done early. If the cord is seriously destroyed, early laminectomy can do no additional harm, and may help by relieving pressure upon parts of the cord not already injured.

If surgical interference is not undertaken, absolute rest should be carried out for a period of two or three weeks. Hyperextension of the spine may be applied, but great care must be used to prevent bed-sores. The patient may be rested upon the chest and abdomen as much as possible, and ice-bags applied to the spine. After the acute symptoms have subsided, the treatment is that of a chronic myelitis.

EMBOLISM AND THROMBOSIS.

Embolism and thrombosis, although rare conditions, may occur in the cord as in other parts of the body. Treatment, which is not promising, must be directed to the vascular system, as in embolism or thrombosis in other parts.

MYELITIS.

Diffuse and disseminated inflammatory lesions not due to direct crushing or contusion of the cord may be spoken of under the term myelitis. Myelitis may be acute or chronic. If the gray matter alone is inflamed, the disease is termed poliomyelitis; if a small vertical extent of the entire cord is affected, transverse myelitis; if an extensive area of both white and gray matter, diffuse myelitis; if a considerable area of gray matter, central myelitis.

ACUTE MYELITIS.

Most cases occur between the ages of 10 and 40 years. Infections and intoxications are the most common cause, and

it may follow almost any infectious disease, or occur in association with purulent foci in other parts of the body. Overexertion and exposure may act as exciting causes. A peculiar form is observed in those who work in compressed air—caisson disease. When the patient passes too rapidly from an atmosphere of compressed air to normal pressure, gas is liberated in the blood, with resultant gas embolism in the blood-vessels, and because of this there are numerous small areas of ischemic softening in the tissues of the cord.

The *symptoms* come on quickly. The most common type is transverse myelitis, and this most frequently attacks the dorsal region of the cord. Usually the first symptom is that of numbness in the feet and legs, soon followed by weakness. If the transverse lesion is complete, there is complete loss of motion and sensation up to a level corresponding to the region controlled by the segment of the cord affected. The reflexes will be abolished in a complete lesion. The muscles of the legs do not atrophy. Retention of urine will be present, and this necessitates catheterization.

If the transverse lesion is in the lumbar enlargement of the cord, there will be loss of control of the sphincters, and rapid wasting of the muscles in the lower extremities. If the cervical enlargement be involved, the wasting of muscles will occur in the arms.

In an acute central myelitis, muscular wasting, paralysis of motion and sensation, and loss of control of the sphincter will be present. All muscles controlled below the upper level of the cord lesion will be affected.

In the majority of cases the myelitis is not complete, and the resulting symptoms are correspondingly confused. Sensation and motion and sphincter control are not entirely lost; and both deep and superficial reflexes are increased in the incomplete cases.

The chief aim of *treatment* is the prevention of bed-sores and cystitis. Great care should be used in catheterization, since the resistance of the bladder to infection is lessened by the trophic disturbance. A water- or air-bed is to be preferred, and absolute rest in bed for weeks is necessary. Diaphoretics and urinary antiseptics may be given. The treatment of the

most favorable form, that due to syphilis, will be considered in the article on this infection. (See p. 689.)

Improvement has been observed after lumbar puncture in some cases, but on the whole outlook is unpromising. Some patients recover partly. The more severely and the more quickly the symptoms arise, the more grave the outlook. Later in the disease, small doses of the iodids, with strychnin and the cautious use of electricity, are indicated.

COMPRESSION OF THE SPINAL CORD.

This affection, also known as compression myelitis, is an interruption in the function of the spinal cord due to slow and continuous pressure. It may be caused by disease of the vertebræ due to tuberculosis, syphilis, or cancer; by new growths developing from the spinal column or membranes; by aneurism of the abdominal or thoracic cavity; or by the growth of cysts within the spinal canal.

The spinal meninges frequently are inflamed at the seat of the lesion, and the cord is thinner than normal at the point of pressure. The nerve elements degenerate at the point of pressure, and secondary degenerations take place in the different tracts of the cord.

The onset of the *symptoms* is slow, and the first symptom noticed is pain of a girdling type, due to irritation of the posterior nerve roots. Later, symptoms of chronic myelitis develop. The motor symptoms, weakness, marked spasticity of muscles, and plus reflexes, are apparent usually before serious sensory disturbance is observed.

If the compression be due to caries of the vertebræ, suitable surgical *treatment* should be applied. If due to syphilis, good results will be obtained by appropriate therapeutic steps, if the case is recognized early. If due to a tumor, the seat of the lesion should be located, and surgery applied to its removal. If due to malignant growth, or aneurism, treatment at best can be only palliative. Proper attention should be given to the care of the skin and bladder, as in myelitis.

CHRONIC MYELITIS.

This affection may be either a sequel of the acute forms, or due to extensions from a meningitis. As a primary affection it is rare, and in such instances is attributable to alcoholism, syphilis, or exposure.

The nervous tissue degenerates, and the connective tissue greatly increases in amount. Secondary degenerations develop, the blood-vessels become thickened, and, macroscopically, the cord is smaller and harder than normal.

The *symptoms* vary with the location and extent of the lesion in the cord. In chronic dorsal myelitis, the first symptoms met with are fatigue in walking, stiffness of the muscles, and paresthesias in the legs. Actual pain is usually present only in a slight degree. All the reflexes are increased, and the muscles more or less spastic. Bladder and rectal disturbances and impotence are present in the late stages of the disease.

The disease lasts for years, and is incurable, so that no *treatment* is efficacious. The patient should be cautioned against overwork and fatigue, and must avoid inclement weather, and, if possible, live in a warm climate. Daily warm bathing is beneficial. A spinal fluid study should be made in all cases, and positive findings of syphilis will call for active antisyphilitic treatment. With negative findings, a carefully watched course of mercury and iodids may be of some benefit. Strychnin should be avoided where much spasticity is present. The nutrition of the muscles may be maintained by electricity and massage.

SYRINGOMYELIA.

Syringomyelia is a rare affection, of obscure etiology. It develops early in life, usually in subjects from twenty to thirty years of age, as a localized gliosis, frequently occupying the central sections of the spinal cord. This new growth extends longitudinally through the cord, outgrows its blood supply, the cells soften, break down, and are absorbed, leaving a cavity formation, surrounded by a wall of proliferated neuroglia of variable thickness.

The course of the *symptoms* is slow but progressive. The usual site of the incipient lesion is the cervical cord, thus ac-

counting for the fact that the first symptoms usually appear in the hands. The picture is that of progressive muscular atrophy of spinal origin. As the lesion extends, the motor columns of the cord are damaged, causing weakness and spasticity of muscles, with increased reflexes. The picture now resembles that of amyotrophic lateral sclerosis, and to these symptoms are added disturbances of sensation. The pain and temperature fibers are destroyed, as they decussate in the gray matter and anterior commissure, while the tactile fibers enter the posterior columns of the cord, and do not decussate until they reach the medulla. This causes dissociation of sensation, or loss of pain and temperature sense, with preservation of the tactile sense. Various symptoms develop as other parts of the cord are involved. The sensory disturbance is distributed according to the spinal cord segments, and not according to the nerve distribution, as in neuritis. Trophic symptoms, such as arthropathies, fragility of the bones, skin lesions, and ulcers may develop. Bed-sores, cystitis, and sphincter paralysis develop late in the course of the disease.

The *treatment* of this affection is purely palliative, and nothing is known to cure or stay its progress. The patient should be protected from extremes of heat and cold, and should not overuse weakened muscles. As a rule, the patient suffers no pain. Trophic lesions, such as ulcers and fractured bones, are to be treated surgically.

ACUTE ASCENDING (LANDRY'S) PARALYSIS.

Landry's disease is an acute progressive paralysis, usually beginning in the legs, and rapidly extending upwards to involve the trunk, upper extremities, and finally the cardiac and respiratory centers. It usually occurs in early adult life. It presents no marked sensory change or changes in the electrical reactions.

It is in many respects analogous to acute poliomyelitis, and is probably due to some infectious agent. In some cases the motor system appears to be so overwhelmed by the toxemia that death results before anatomical changes develop in the nerve tissue.

The *treatment* is wholly palliative. The patient must have absolute rest in bed, with care of the bowels and bladder. There is no treatment known to check the process, whatever it may be. Death ensues when the medulla is reached. Some cases recover, but there is no special treatment which brings about their recovery. Supporting measures should, of course, be used.

MULTIPLE SCLEROSIS.

An embryologic defect, tending toward the proliferation of the neurogliar tissue, is probably the essential cause of most cases of multiple sclerosis. The acute infectious diseases, intoxications such as mercury and carbon monoxid, grave psychic disturbances, and severe trauma, all have been assigned as causal factors.

The disease is characterized by multiple lesions indiscriminately distributed throughout the brain and spinal cord; these foci, macroscopically, can be distinguished from the surrounding tissue. Microscopically, many axis cylinders are seen to be preserved, but very few retain their medullary sheath. The neurogliar tissue is increased, and does not show any tendency toward softening. Secondary degenerations are absent, and ganglion cells, even in the center of the foci, are not injured.

The cardinal *symptoms* of a fully developed case are a coarse intention tremor, nystagmus, and scanning speech. To these may be added optic atrophy, especially marked on the temporal side of the discs, and the symptoms of a spastic paraplegia (spastic and weak muscles, plus knee-jerks, Babinski sign, and ankle clonus).

No *treatment* is known that can cure the disease. We can only hope that the particular case under treatment is one destined to undergo a marked remission. It is very important that the patient be thoroughly rested and nourished, by forced feeding, if necessary. If anemic, hematinics like iron and arsenic may be used. The disease is not painful, and does not interfere with the functions of the various organs of the body. Physical overexertion, undernutrition, and psychic excitement are to be avoided. The use of alcohol and tobacco should be interdicted.

THE MUSCULAR DYSTROPHIES.

The muscular dystrophies, or myopathies, are probably due to an intrinsic disease of the muscles themselves; or, possibly, disease of some of the glands of internal secretion, *e.g.*, the thymus, may play a causative rôle. They are characterized by atrophy beginning in certain groups of muscles, and often associated with a pseudo-hypertrophy in the same muscle or in other muscles. In the very early stages a true hypertrophy of the muscle fibers may be present. In others there is a proliferation of muscle nuclei and longitudinal splitting of the fibers. There is an increase of connective tissue, which takes the place of the atrophied muscle fibers. Extensive deposits of fat take place in the connective tissue in the pseudohypertrophic form.

The only known etiologic factor is heredity, and the disease ordinarily occurs in several generations of a family; it begins in childhood, as a rule, but some cases appear in early adult life.

The *symptoms* vary greatly, and a number of clinical types have been described, depending on the part of the body in which the muscles are first attacked, the age of onset, and the occurrence of hypertrophy.

They may be classed as the leg type, the shoulder-girdle type, and the face type, between which three forms there is no sharp line of division. The same family may present each of the different types. In all cases sensory symptoms and fibrillary tremors are wanting, and there is a diminution of reflexes. The parts are cold, and deformities often develop. The leg type especially presents the form of muscular pseudohypertrophy. It usually develops in a child under the age of 10 years, the patient presenting enlarged muscles, especially of the calf, thighs, and buttocks, with corresponding weakness and awkwardness of gait. One characteristic sign of the disease is the way in which the child arises from the floor. He first gets on all fours, and gradually raises his body by supporting himself with his hands on his knees and thighs, thus "climbing up his legs" when completing the act of rising.

The shoulder-girdle type presents the disease in the large muscles of the shoulder and chest, such as the deltoid, pec-

torals, biceps, triceps, and supra- and infra-spinati. Pseudo-hypertrophy may or may not be present; usually it occurs later in life than either the face or the leg type.

The face type usually begins early in childhood, and the muscles of the face are first affected, the eye muscles and those of mastication escaping.

No cure of myopathy of the types mentioned is known. The disease advances slowly, and the patient may live for many years.

Although *treatment* is of no avail, members of a family subject to muscular dystrophy should be advised of the dangers of a hereditary taint affecting the succeeding generation. The children should have the best of care and surroundings, and be protected from excessive fatigue of all kinds. The treatment of the disease itself, although unsatisfactory, calls for massage and electricity, as well as for the use of strychnin. The extracts of the thymus and pituitary glands have been thought to do good in some cases. For the contractures incident to this affection surgery may be resorted to, and tenotomy may relieve the resulting deformities.

PROGRESSIVE NEURITIC MUSCULAR ATROPHY.

This form of atrophy usually begins in the muscles of the feet, and extends upward. It usually arises before the age of 20, and is more common in males than in females. It is a hereditary disease.

The nerve fibers are found to be degenerated, and an excess of connective tissue, with proliferation of cells, is found in the neurilemma. The muscles show atrophy of their fibers, with proliferation of the connective tissue.

The *symptoms* consist of weakness of the muscles of the foot, peroneal, and anterior tibial regions of early development, and gradually extending to other muscles of both the lower and upper extremities. The reflexes are diminished, and sensory disturbances in the form of pain, tenderness to pressure, and paresthesia are present. The affected limbs are likely to be cold and cyanotic.

There is no prospect of recovery, although the progress of the disease may be slow.

There is no radical *treatment* for this type of atrophy, but the use of electricity and massage is indicated as a palliative measure. Deformities may result, which call for tenotomies or braces. The weakened muscles should not be overfatigued.

SYPHILIS OF THE NERVOUS SYSTEM.

Syphilis of the nervous system is an infectious disease due to the *Treponema pallidum*. In former years it was customary to speak of two groups of syphilitic disease of the nervous system, the first being characterized by specific or gummatous inflammatory lesions attacking the nerve tissue and its coverings by way of the blood-vessels; the second group was spoken of as parasyphilitic disease, characterized by degeneration of the nerve tissue, and believed to be due indirectly to syphilis, in from 60 to 90 per cent. of the cases. At the present time both groups are recognized as active syphilis, due to the same infectious agent.

However, while the underlying cause is the same in all forms of lues of the nervous system, we are obliged to recognize two groups as different and distinct clinical affections. From the standpoint of prognosis and treatment, this distinction is important. In the first group the infection attacks the nervous system by way of the interstitial tissues, and it may be spoken of as the interstitial form, or cerebrospinal syphilis of the vessels and membranes. Because of the formation of gummatous deposits, and the infiltration of vessels and membranes, the first group also may be conveniently spoken of as the exudative form. In the second group we recognize that the nerve tissue itself is attacked, and it may be referred to as the parenchymatous form, including the diseases, paresis and tabes dorsalis.

One characteristic feature of interstitial nervous syphilis is its multiplicity of *symptoms*. In one case the cranial nerves especially may suffer, in another the brain, in another the spinal cord, and in still another, the entire cerebrospinal axis. In brain syphilis, a common symptom is headache, usually worse at night, and preventing sleep; somnolence during the

day time, mental dullness, irritability, and at times stupor or delirium also occur. Some cases develop convulsions, either focal or general in character, and some apoplexy, generally due to thrombosis. Various cranial nerves may be attacked, especially the sixth and the third. Optic neuritis and choked disc may occur.

The syndrome of spinal syphilis as described by Erb is the most common. This is characterized by early bladder disturbance, usually a slowness in starting the flow of urine, plus reflexes, and spastic gait, with comparatively little spasticity of the leg muscles when the patient is seated. Paresthesias and some irregular areas of sensory loss may be present. Parts of the cord other than the lateral columns may be attacked. Implication of the posterior columns and nerve roots may cause absent knee-jerks, and lightning pains suggesting tabes. If the lesion should interfere seriously with the central part of the cord, symptoms of dissociation of sensation may develop. If the anterior horns are affected, atrophy takes place in corresponding muscles, thus explaining the occurrence of some cases of chronic progressive muscular atrophy and chronic anterior poliomyelitis.

The *diagnosis* of interstitial nervous syphilis rests upon the multiplicity and irregularity of the symptoms, which can only be accounted for by multiple lesions, by the fact that the pains are worse at night, and by the study of the blood and spinal fluid. The blood Wassermann test should be positive. If the disease has attacked the membranes of the cord and brain, the spinal fluid Wassermann test should be positive, and the cell-count high.

Tabes usually presents as early symptoms sharp shooting pains in various parts of the body, such as the legs, upper abdomen, bladder or rectum, spoken of as crises; sluggishness in the reaction of the pupils to light; and diminished or lost reflexes. Later the Argyll Robertson pupil develops, and also the ataxic gait and station, bladder symptoms, and trophic disturbances, such as the Charcot joint. The Wassermann reaction in the blood may be absent, but commonly it is strongly positive in the spinal fluid, together with increased globulin and a cell-count varying from 20 to a very large

number of lymphocytes, depending upon the severity and activity of the disease at the time of examination.

Paresis presents both mental and physical symptoms. The characteristic mental symptom is mental failure or dementia. The patient early in the disease complains of ill health, and his early symptoms may strongly suggest neurasthenia. Later he becomes irritable, forgetful, and shows loss of judgment. Some cases become depressed, and may show delusions of self-blame resembling melancholia. Others become exalted, with grandiose delusions, which resemble mania. Other cases simply show dementia with no delusions. Among the important physical signs are disturbances of the pupils, such as the Argyll Robertson pupil, irregular pupils, and unequal pupils which do not respond to light; diminished, exaggerated, or unequal knee-jerks; a fine tremor of the lips, tongue, and hands; a slurring, drawling, hesitating speech, and an irregular, shuffling, or ataxic gait. Both the blood and spinal fluid show a positive Wassermann reaction, the latter having also increased globulin, and a lymphocyte-count rarely above 100 cells per cubic millimeter.

The *diagnosis* rests upon the mental and physical signs, especially the pupillary signs and the positive Wassermann reaction in both the blood and spinal fluid, with the presence of increased globulin and lymphocytes in the latter.

The *prognosis* as to the cure of nervous syphilis should be guarded in the interstitial form, and is distinctly unfavorable in the parenchymatous variety.

TREATMENT.

In no case of syphilis in the early stages should we counsel a discontinuance of treatment, even though the blood Wassermann be negative, so long as the spinal fluid is positive. Many cases of syphilis show nervous symptoms a few months after the occurrence of the chancre, and in others an examination of the spinal fluid reveals a positive Wassermann when possibly no nervous symptoms are present. Treatment continued and directed with a knowledge of the condition of the spinal fluid rather than of the blood will probably prevent severe cases of nervous syphilis in later years.

Syphilis is one disease in whose management we resort largely to drugs, which here have an almost specific effect. The most valuable drugs are mercury, arsenic, and iodin in various combinations. The treatment of interstitial or cerebrospinal syphilis, to be satisfactory, depends upon an early diagnosis. If a large gumma has formed in the brain, drugs will not remove it, and the case may have to be treated surgically, as in other brain tumors. Again, if a thrombus due to syphilis forms in a blood-vessel of the brain or cord, there will be a more or less permanent destruction of nerve tissue that drugs will not remedy. With an early diagnosis, such untoward results are less frequent. Arsenic, in the form of salvarsan ("606") or similar preparations, should be given intravenously. If it is thought best not to use salvarsan, the patient should be put on mercury. This can be given hypodermically in the form of bichlorid, calomel, or gray oil, but the most effective method of administration in nervous syphilis appears to be by inunction. The ordinary 50 per cent. blue ointment or oleate of mercury can be used, from 1 to as much as 4 drams (3.9 to 15.5 Gms.) being well rubbed into the skin each day. It is essential to place the patient under the influence of the remedy as rapidly as possible. If the brain is severely attacked, it is well at the same time to give rapidly increasing doses of some iodin preparation, preferably sodium iodid. If the patient suffers from a mild attack, it is well to use the mercury alone for a period of about six weeks, this to be followed by a course of gradually increasing doses of the iodids for the same length of time. These courses of treatment should be repeated at intervals, provided that symptoms of active disease and a positive Wassermann in the spinal fluid are still present.

In tabes and paresis or parenchymatous nervous syphilis, the above method of treatment does little good. For some reason not explained, drugs which act on syphilis of the exudative form, do not produce any such result when the spirochete has invaded the nerve tissue itself. It has been found that with either mercury or salvarsan circulating in the blood practically none can be found in the cerebrospinal fluid. Efforts have been made to devise some method of medicating the brain and cord directly by way of the subarachnoid space.

The method of Swift and Ellis has proved to be one of the most useful. (*Cf.* Syphilis, p. 84.) By this method the patient is first given an intravenous injection of salvarsan or neosalvarsan. After an interval of from twenty minutes to one hour, blood is drawn from the vein. The blood is allowed to stand overnight; the serum is drawn off, and then thoroughly centrifugalated; after this, it is inactivated at a temperature of 56° C. (132.8° F.). Ten to 20 mils (2.7 to 5.4 f3), diluted with an equal or double amount of salt solution, is then injected intraspinously. This is usually given twenty-four hours after the intravenous injection. The patient is placed in lateral decubitus in bed, with the knees well drawn up, and the head bent forward upon the chest. A lumbar puncture is then made, and the spinal fluid allowed to drain off to the amount of 40 mils (10.7 f3) or more. In most cases the dural sac can be drained of all the fluid that will flow. The serum, warmed to body temperature, is then allowed to enter, preferably by gravity, through the puncture needle into the dural sac. The needle is then withdrawn, the patient turned on his back without a pillow, and the foot of the bed raised. He is kept in this position for three hours, and then allowed a more comfortable position. This treatment may be repeated in two weeks, in most cases of tabes or paresis. The reaction in tabes may be so severe, with such severe crises resulting, that a longer period of time between the treatments may be necessary. The patient should remain in bed for from one to three days after a treatment.

In Ogilvie's method 40 or 50 mils (10.7 or 13.4 f3) of blood are taken from the patient, and the serum centrifugalated. One-fourth milligram of salvarsan, dissolved and neutralized in the usual way, is added to the serum; this mixture is incubated at body temperature for one hour, and then inactivated for one-half hour at 56° C. (132.8° F.). About 10 mils (2.7 f3) of this salvarsanized serum are injected into the dural sac, by the technic just described.

Another method, which has not been much followed in paresis, is to inject the serosalvarsan, as prepared by the Swift-Ellis method, directly beneath the intracranial dura. Previous to the injection, intracranial pressure is reduced by lumbar puncture. The great objection to this treatment is the opera-

tive procedure necessary to its application. Byrnes advises the use of bichlorid of mercury, which gives good results, especially in tabes. Many of the foregoing methods do good in tabes, and in some cases the results are striking. The more early the diagnosis is made, and the more acute the inflammatory lesions, as indicated by a high cell-count, the more striking are the results.

In paresis the results are not so encouraging. If the case is seen early, and the diagnosis made while the patient presents only neurasthenoid symptoms, with few physical signs, the chances for inducing a more or less prolonged remission are good.

Too much stress cannot be laid on the fact that mercury should not be forgotten in treating all forms of nervous syphilis. In the light of the knowledge that cases of tabes or paresis, receiving either mercury or arsenic by way of the skin, mouth, or blood-vessels, rarely or never show these drugs in the cerebrospinal fluid, we began three years ago to treat these patients at the Jefferson Hospital Nervous Clinic, by draining off the cerebrospinal fluid at intervals, with the idea that, by lowering the pressure within the cerebrospinal canal, we could encourage the diffusion of these drugs from the blood of the capillaries into the cerebrospinal fluid. In carrying out this treatment, we have used mercury. Our plan has been to give the patient suffering from either tabes or paresis, mercury by inunction, and to drain the cerebrospinal fluid once every week or two weeks.

We place the patient in bed, drain by a lumbar puncture with a Quincke needle all the fluid that will flow, usually from 15 to 60 mils (4 to 16 f3). We keep him in bed overnight, and allow him to leave the hospital the following morning. We have not been able to demonstrate mercury in the cerebrospinal fluid. We have seen cases of tabes improve, both from the clinical and laboratory standpoint, just as much when treated by mercury, with systematic drainage of the cerebrospinal fluid, as when treated by the Swift-Ellis method with salvarsan. We have seen remissions and improvement in cases of paresis, treated early. We find that the success of the method depends on the ability of the patient to use inunctions to the point of ptyalization. His teeth must be carefully

looked after, and the mercury inunction pushed to the limit of tolerance, and kept at this point for at least three months. After drainage of the cerebrospinal fluid a number of times, the patient improves in general health, regains his weight, and has less pain. The advantage of this treatment is the simplicity of the technic, and usually the absence of any severe reaction. Some patients, especially those with cerebrospinal syphilis, complain of severe headache after drainage. In such cases it is necessary to keep the patient quiet until the headache subsides. The more skill employed, and the more ease with which the lumbar puncture is done, the less is the liability to headache.

With the present state of our knowledge of tabes and paresis the ideal method seems to be as follows:

1. Early diagnosis before serious destruction of nerve tissue has taken place.

2. A series of serosalvarsan treatments according to the Swift-Ellis method, the number of these treatments to be determined by Wassermann tests of both blood and spinal fluid.

3. Following the serosalvarsan treatment a thorough course of mercury by inunction, with the drainage of the cerebrospinal fluid once weekly, for a period of at least three months.

4. Recognition of the fact that nervous syphilis is incurable with the means at present at our command, that the patient must be kept under observation, and that the treatment must be repeated as often as the serological findings indicate.

The older methods of treatment in tabes and paresis are to be employed as indicated. Whenever possible the patient should be given a thorough rest treatment—rest in bed, full feeding and massage—in order to bring the resistance to the highest possible point. These patients should never lead strenuous lives, with an excessive amount of hard work. When the mental changes become so marked that the paretic no longer can be at large, confinement in an institution is necessary. The treatment here is simply protective, making his last days as comfortable as possible under the circumstances.

Some cases of tabes suffer from severe pains in spite of all specific treatment, and if this be so resort must be had to

drugs, such as acetphenetidin and aspirin, and in severe cases to morphin, although an opiate is to be withheld as long as possible. The pains frequently are relieved by drainage of the spinal fluid, and this should be tried before morphin is used. The bladder must be watched for infection, and treated by irrigation if there be much pus, and drugs such as atropin, strychnin, and urotropin given internally. For severe ataxia the persistent use of Frenkel's method of exercise is indicated.

DISEASES OF THE PERIPHERAL NERVES.

PRESSURE PALSY.

Long continued pressure upon a nerve may cause paralysis of the muscles supplied by it. The musculospiral is the nerve most frequently affected because of the not infrequent habit of sleeping with the head resting upon the arm. Not uncommonly such a palsy occurs during the heavy sleep produced by alcohol. Other nerves, such as the sciatic, ulnar, or anterior tibial likewise may suffer from pressure palsy.

The *diagnosis* rests largely upon the history of the case and the absence of pain and tenderness in the affected nerve, a fact which distinguishes the affection from neuritis. In musculospiral palsy, lead poisoning may suggest itself, and here not only the history of the case, but the fact that lead palsy is bilateral, serve to make the distinction.

Rest of the paralyzed muscles, massage and electricity constitute the *treatment*. Strychnin may be given, but it is of little use. The prognosis is almost uniformly good; practically all cases recover in a few weeks.

NEURITIS.

An inflammation of a single nerve trunk is spoken of as a local neuritis; when a number of nerves are affected, the term multiple neuritis is applied. Neuritis may be acute or chronic and the acute form may pass into the chronic.

The inflammatory process may be interstitial, in which case the connective tissue is the primary seat of the lesion; or it may be parenchymatous, in which case the nerve tissue

itself is primarily affected. In the interstitial form the peri- and endo- neurium are the first to suffer, and the changes in the nerve tissue are secondary to those in the connective tissue. In the parenchymatous form the nerve trunks are firmer and grayer than normal. The myelin is segmented and divided into drops and granules, and the axis cylinders become granular and finally disappear. The nuclei on the sheath of Schwann proliferate and the nerve trunk finally becomes a fibrous cord. Secondary changes are sometimes found in the anterior horn cells.

LOCAL NEURITIS.

Exposure to cold, extension of inflammation from surrounding tissues, and trauma are the most common causes of a local neuritis. Localized neuritis is usually interstitial.

The *symptoms* relate chiefly to pain along the course of the nerve and in the part supplied by the nerve. This pain is of a dull, boring character, and made worse by movement of the affected part. The nerve is also tender to pressure. At times herpes, edema, or redness of the skin over the nerve are present. The muscles supplied by the nerve are weak and flaccid, and more or less atrophy may make its appearance. Electrical changes also may be noted. These vary from a quantitative change to complete RD. Various sensory disturbances, paresthesia, hyperesthesia, and at times total anesthesia may be present.

The duration of the symptoms depends among other things upon the severity and the extent of the affection. Accordingly it may vary from a few weeks to several years.

The *diagnosis* rests especially upon the presence of pain and tenderness of the nerve trunk, or in the smaller branches in the skin and muscles. The pain of neuralgia is commonly paroxysmal and the nerve trunk is not tender to pressure except for a short while after a paroxysm of pain; and then the nerve is sensitive rather than sore or tender. In spinal root irritation, again, the pain is girdle-like in distribution and the nerve trunks are not tender to pressure.

TREATMENT.

The part of the body in which the inflamed nerve is situated should be put at rest, and this rest should be as nearly absolute as possible. The earlier the diagnosis is made and rest applied, the more rapidly the case recovers. If, for instance, the inflamed nerve is in the arm, it is preferable early in the case to put the arm on a splint. Later, as the pain subsides, the arm may be carried in a sling. The part affected may be wrapped in cotton and lightly bandaged or some cooling lotion may be applied, such as lead water and laudanum.

For the relief of pain, the coal-tar products and salicylates may be given, or, if the pain is very severe, opiates are demanded. The constant galvanic current may be applied, the positive pole being placed over the inflamed nerve for ten or fifteen minutes daily. Little dependence, however, can be placed upon this expedient. After the acute symptoms have subsided, massage, electricity, and strychnin are indicated. The use of dry heat by means of a dry hot air apparatus is often of marked benefit. The massage should be applied with the view of maintaining the nutrition of the affected muscles and nerves; electricity may likewise be used to stimulate the affected nerve and muscles. That form of current should be used which trial indicates as producing the best responses in the muscles with the least discomfort to the patient.

MULTIPLE NEURITIS.

Multiple neuritis is probably always due to some poison circulating in the blood. The inflammation is usually of the parenchymatous form. Among the various causes are:—

1. Poisons taken into the body from without, of which alcohol, carbon monoxid, carbon bisulphid, lead, arsenic, and mercury are examples.

2. Poisons generated within the body in the form of toxins of infectious diseases such as influenza, typhoid fever, diphtheria, and pneumonia, septicemia, syphilis, and tuberculosis. Among the multiple neuritides due to infection, beriberi in all probability also should be included, although its exact causal relationship has not been positively determined.

3. Poisons generated within the body because of faulty metabolism, such as in gout and diabetes, or those which have their origin in pregnancy and the puerperal state.

4. Dyscrasic blood conditions, such as chlorosis, marasmus, cancer, or cachexia from any cause.

The *symptoms* differ somewhat in different cases. In some the sensory fibers suffer chiefly, while in others the motor fibers are especially affected, and in others still both sensory and motor fibers are affected. Again, in some forms certain nerves especially are attacked such as the musculospirals in plumbism, the anterior tibials in alcoholics, and the nerves supplying the soft palate in diphtheria.

The general symptoms of multiple neuritis are similar to those described under local neuritis. Pain and tenderness along the course of nerve trunks, diminished or lost reflexes, wasting of muscles, and sensory disturbances are present in varying degree. In many cases, as might be inferred from what has been just stated, little or no pain or tenderness can be elicited, as in the multiple neuritis of lead poisoning and of diphtheria. In others, again, pain and tenderness may be very pronounced, as in many cases of alcoholic multiple neuritis. Further, pain and tenderness are frequently absent or but slightly marked in the larger nerve trunks, but pronounced in the smaller branches. This is not infrequently the case in alcoholic multiple neuritis. Here, indeed, tenderness is commonly elicited by forcibly grasping the forearm above the wrist or the leg above the ankle.

The palsies of multiple neuritis are in given cases quite characteristic, as instanced by the paralysis of the palate and nasal regurgitation of liquids in diphtheria, the double wrist-drop of lead poisoning, and both the double wrist-drop and foot-drop of alcoholic multiple neuritis.

Finally, it is important to add that in multiple neuritis the sphincters are never affected. This point enables us at once to differentiate this affection from diseases involving the cord. The iris likewise escapes, and its reflexes remain unimpaired.

TREATMENT.

In every case the cause should if possible be determined. If the cause can be discovered and eliminated, as in alcoholic

multiple neuritis, the probability of recovery is greatly increased. In all cases rest in bed and full rest measures should be applied (see p. 584). A patient with neuritis attended by severe pain will naturally seek the bed, but others, as in those following diphtheria, may suffer little pain and yet be greatly in need of rest in bed. The limbs should be supported upon soft pillows, or be wrapped in cotton and lightly bandaged. With absolute rest in bed and the proper placing of the limbs there will be little need of giving pain-relieving medicines.

The etiology of a given case, of course, influences the treatment. If it is due to some poison such as alcohol or lead, the elimination of the latter is the first step. If due to a diathetic factor, as gout, the diet must be regulated, and antigoutic remedies given. After the pain has subsided, or in a measure been relieved, massage and passive movements may be instituted. Later on electricity may be employed to exercise the muscles. Strychnin also is useful but should not be given early; at first small doses, and later larger doses, may be administered. Care should be taken by means of passive movements, and at times by mechanical appliances, to prevent deformities which sometimes result from contractures.

NEURALGIA.

Neuralgia is characterized by pain along the course of the nerve trunks. The pain is intermittent and usually severe. The nerve trunk is not tender to pressure as in neuritis, but often reveals discrete painful points.

The causes of neuralgia are very numerous. Diathetic causes, toxins, vices of nutrition, and inherited tendencies play here a rôle. The various poisons which are responsible for the production of neuritis may be factors in the production of neuralgia; in addition to these causes there are various peripheral irritations, of which eye strain, nasal and sinus disease, and carious teeth are examples. Furthermore, it is a disease of adults and not of children.

The important *symptom* of neuralgia is pain, which is paroxysmal, sharp, shooting, or burning in character. Between the paroxysms a dull pain may persist. The pain is increased or brought on by irritation, as from cold, heat, or

motion of the affected part. Hyperesthesia is frequently present over the area of nerve distribution.

The *diagnosis* rests upon the character of the pain and the absence of the symptoms of other affections.

TREATMENT.

The general principles of rest and feeding—the stimulation of nutrition—apply in this condition, as in neuritis. Very often with the improvement in the general health of the patient the pain disappears. The cause of the pain should of course be sought for and removed, if possible. Sources of peripheral irritation must be borne in mind. Especially must infected teeth, nasal, throat, and ear conditions be looked into. If the affection be the result of arteriosclerosis little or nothing can be accomplished, as a rule. Great attention should be given to the gastro-intestinal tract, since constipation may be productive of neuralgia; indeed, the good effect of repeated doses of castor oil noted in some cases is probably to be accounted for by its effect in overcoming constipation. Some attacks of neuralgia come on periodically, and are relieved by full doses of quinin. The salicylates are useful in many cases. Every effort should be made to avoid the use of opium, because of the obvious danger of establishing a habit. Strychnin in increasing doses is very valuable, especially in trigeminal neuralgia. Here its administration in increasing and finally in massive doses is often followed by brilliant results. Electricity is of little use in severe cases.

DISEASES OF SPECIAL NERVES.

Diseases of the cranial nerves are usually associated with diseases affecting the brain and its membranes, and their treatment is that of these diseases. The special exceptions to this rule are the fifth and seventh cranial nerve.

THE FIFTH NERVE.

The fifth, or trigeminal nerve, consists of two portions, a motor and a sensory. The motor portion controls the muscles of mastication and the tensor tympani. The sensory

portion supplies the face and head as far back as the occiput, the conjunctivæ and mucous membranes of the mouth, tongue, upper pharynx, teeth, salivary, and lachrymal glands. Paralysis of the fifth nerve is of rare occurrence; it does not result from exposure to cold, as does facial palsy. Syphilis, hemorrhage, trauma of the skull, and tumor may be a cause; bilateral hysterical palsy may be met with. Treatment depends, of course, upon the cause and is based upon general principles.

Neuralgia of the fifth nerve is characterized by pain in one or more of its divisions. Quite commonly it is accompanied by tenderness to pressure over the infra-orbital or mental foramina or over the supra-orbital notch; the so-called points of Valleix. Not infrequently the paroxysms of pain are accompanied by spasms or twitchings of the facial muscles, the affection being then termed *tic douloureux.*

TREATMENT.

As already insisted upon, great attention should be paid to building up the general health of the patient; this in some cases will cause the attacks to become less severe. In addition, all sources of peripheral irritation should be carefully investigated. Salicylates and coal-tar products may be used to relieve the pain. Strychnin in gradually increasing doses often does good. The positive pole of the constant galvanic current may be tried.

Not infrequently surgical procedures must be resorted to. Alcohol may be injected into the infra-orbital, mental or supra-orbital foramina. This frequently gives relief for many months at a time. In more obstinate cases, the injections may be made into the foramina of exit at the base of the skull. Again the branches of the nerve may be resected, but the relief obtained from this procedure is not permanent. If the resection is made as near the exit of the nerve from the skull as possible and is continued near to its termination in the skin—according to the method of the late W. J. Roe—the pain may not return. The most radical procedure consists of the removal of the Gasserian ganglion, or, better still, the evulsion of the sensory root of the trigeminal. This, of course, necessitates a severe operation.

THE SEVENTH NERVE.

A lesion involving the seventh nerve produces paralysis in some or in all of the muscles supplied. The lesion may affect either the central or peripheral neuron, the palsy being spoken of, accordingly, as central or peripheral facial paralysis. A lesion may attack the central neuron anywhere in its course through the brain; from its origin in the motor cells of the precentral convolution to its end at the facial nucleus. The peripheral neuron may be attacked at its origin in the facial nerve nucleus or elsewhere in the course of the seventh nerve. The palsy resulting from a lesion of the peripheral neuron is known as Bell's palsy. The nerve may be attacked within the skull, within the fallopian canal, or external to the stylo-mastoid foramen.

Central palsies are due to some brain lesions, such as hemorrhage, tumor, or abscess affecting the internal capsule. Consequently central facial palsy is commonly associated with an ipsolateral hemiplegia.

Peripheral palsies may be due to disease of the nucleus, to the exudation of a meningitis, to a tumor or fracture at the base of the skull, to a neuritis due to trauma, exposure to cold, or to an extension of inflammation from middle-ear disease. The most common causes of Bell's palsy are middle-ear inflammation and exposure to cold.

A central palsy usually does not affect the muscles of the upper part of the face, and the *symptoms* relate to a more extensive underlying condition, a hemiplegia. The muscles are not wasted, and the electric reactions are normal. A peripheral palsy affects all of the muscles of the side of the face. The eye cannot be closed, the forehead cannot be wrinkled, the lower half of the face is flattened and flaccid, while the angle of the mouth is drawn to the opposite, the sound, side. Subsequently the muscles may undergo atrophy, and changes in their reaction to the electric current will be noted. Later secondary contracture of the paralyzed muscles may ensue.

There is no loss of sensation, although there may be some pain, usually slight in character, about the ear and mastoid process. If the lesion is intracranial, the eighth nerve also is usually affected, causing deafness; if the nerve is at-

tacked in the fallopian canal, the sense of taste is lost in the anterior two-thirds of the tongue, due to implication of the chorda tympani; if the lesion is extracranial, neither taste nor hearing is interfered with.

The differentiation must first be made between a central and a peripheral palsy. This usually presents no difficulties; the fact that the palsy affects all of the muscles in the facial supply readily determines the *diagnosis*.

TREATMENT.

The treatment of a central palsy is that of the disease of which it is a part, usually that of hemiplegia. The treatment of a peripheral palsy is satisfactory in most cases. The cases which show a complete RD on electric examination are the ones which yield most slowly to treatment and may require care for a year or more. In some cases of otitis media the nerve is so damaged that it does not recover, and in such instances an anastomosis of the facial nerve with the hypoglossal or spinal accessory may be considered. If the electric examination shows only a quantitative loss, the patient usually recovers quickly; possibly in a few weeks, with little treatment. In early extracranial cases, due to exposure to cold, a blister may be applied back of the ear, and full doses of salicylates and a brisk purge are indicated. The salicylates are of benefit, even although a distinct rheumatic factor cannot be determined. Later, small doses of the iodids may be given. After the acute symptoms have subsided strychnin may be given, $\frac{1}{60}$ to $\frac{1}{40}$ grain (0.001 to 0.0015 Gm.) three or four times daily. After about ten days the electric current may be employed to stimulate the paralyzed muscles. If the case is severe the interrupted galvanic current should be used, and the best contractions are usually obtained by applying the anode or positive pole to the muscles. If the case is mild, the slowly interrupted faradic may suffice. The electric treatment should not be continued too long at a sitting for fear of exhausting the weakened muscles. Massage is of more importance than electricity, and should be used from the beginning to the end of the treatment. The patient can usually be taught to apply the massage himself; he should be instructed to rub the face with a circular motion, at the same

time raising between the thumb and fingers the muscles and tissues of the face. The persistent use of massage tends to prevent, or at least to lessen, the contractures which may develop in the paralyzed side.

BRACHIAL PLEXUS.

Through the brachial plexus the muscles and skin about the shoulder, arm, forearm and hand are innervated. It is frequently the seat of injury by direct violence, or by pressure exerted by a dislocated humerus. In complete paralysis the lesion is usually due to an injury which ruptures the nerves in the plexus, or more rarely, tears the nerve-roots from the spinal cord. In complete lesions, motion, sensation, and nutrition of the entire arm and shoulder are affected.

A partial plexus paralysis, as originally described by Erb, regularly implicates the deltoid, biceps, brachialis anticus, and supinator longus. In some cases the supinator brevis, the infraspinatus, and the subscapular are paralyzed, and in examples of this sort the lesion is supposed to affect the fifth and sixth cervical nerves or the upper trunk of the brachial plexus. Trauma is the usual cause of such a paralysis. The so-called obstetric paralysis is supposed to be due to stretching of, or pressure upon, this part of the plexus during birth.

Klumpke's paralysis implicates the eighth cervical and first thoracic nerves, and the symptoms produced are manifest in the small muscles of the hand and the flexors of the forearm. At the same time, due to disease of the ramus communicans, there is a paralysis of the sympathetic upon the same side, evidenced especially by retraction of the eyeball and contraction of the pupil.

The outlook in these forms of paralysis depends on the extent of the original lesion. In total paralysis, which usually means a rupture of nerve-trunks, the outlook is naturally very unfavorable. In the upper arm type, especially in obstetric paralysis, there is a tendency toward improvement, the extent of which, of course, depends upon the severity of the injury. The same rule applies to paralysis of the lower arm type. If the lesions are due to a new growth or to any other

progressively increasing cause, the outlook is, of course, unfavorable.

Both neuritis and neuralgia of the brachial plexus, affecting one or all branches of the plexus, are not uncommon. Trauma not so severe as to produce paralysis may be a cause. Rheumatism is a not infrequent factor. Neuralgia may be due to any of its various causes, but in such cases the possibility of a direct irritation of the cervical nerve-roots must always be borne in mind.

The diagnosis of a neuritis depends upon the presence of pain upon pressure along the nerve-trunks, pain upon motion of the shoulder and disturbance of motion, sensation, and nutrition of the parts supplied. The diagnosis of neuralgia depends upon the presence of pain with the absence of the signs of neuritis.

TREATMENT.

If a complete RD in the muscles, with total loss of motion and sensation, is present and continuous, the evidence tends to show that nerve-trunks are completely severed and surgical intervention offers the only possible remedy. In other cases the use of massage and electricity and all measures which help the nutrition of the nerves and muscles are indicated.

In brachial neuritis an early diagnosis and institution of treatment are important. The first essential is complete rest, preferably by placing the patient in bed, and properly supporting the arm. In other instances the arm may be placed in a sling or bandaged to the side of the chest. Among drugs the salicylates are of benefit in most cases. The local application of heat, either moist or dry, is of decided benefit. Electricity in the form of the continuous galvanic current applied with the positive pole to the painful nerves, may also give some relief. As soon as the acute symptoms have subsided the shoulder and arm should be treated by massage. Benefit may be obtained by rubbing in an ichthyol ointment, although it is probable that the rubbing and not the ichthyol is of benefit. Electricity also should now be applied. The arm should be supported by a sling until the symptoms have largely subsided, inasmuch as the pulling and stretching of the nerve

trunks which ensue in moving the arm and shoulder may irritate and again produce pain and tenderness in the affected parts. Full feeding, general tonics, iron and strychnin will prove useful in the later stages.

The treatment of a brachial neuralgia follows the general principles already considered. Rest is essential, and the more complete the rest, the less frequent the paroxysms of pain. If due to a toxic cause, such as malaria, a rheumatic or gouty diathesis, drugs appropriate to such conditions are indicated. A thorough search for peripheral causes of irritation should always be made.

AFFECTIONS OF INDIVIDUAL ARM NERVES.

The median, ulnar, musculospiral, and circumflex nerves are frequently subject to injury. The musculospiral, because of its location, is most frequently injured by pressure. This usually is brought about by the patient sleeping with his head on his arm while under the influence of alcohol. The outlook is good in these forms of pressure palsy, the patient recovering in a few weeks under treatment with massage and electricity.

If in a wound of the arm any of these nerve-trunks are severed they must be treated from the surgical standpoint, followed by a prolonged course of electricity and massage.

DORSAL NERVES.

The most common affection of the dorsal nerves is intercostal neuralgia, the general causes and treatment of which have already been outlined. Local neuritis may arise, followed by herpes, which usually gives much pain and discomfort. General measures to combat any gouty or alloxuric diathesis are indicated. The herpes must be treated locally, with the idea of keeping the skin dry, and, as far as possible, to prevent infection of the vesicles.

LUMBAR AND SACRAL NERVES.

Lesions of the lumbar and sacral nerves and plexuses are not as common as those of the brachial plexus. They may

be affected in disease of the vertebræ, by various injuries, by growths within the spinal canal, and by lesions in the pelvis or in the course of the nerves. The results of the lesions are similar to those noted elsewhere. In a general neuritis these nerves may participate in the general inflammatory process.

The most important affection of the nerves of the lower extremity is sciatica. The sciatic nerve may be the seat of an intense neuralgia. The pain may be diathetic in origin, or due to some irritation in the pelvis, such as a local pressure. Very frequently a neuritis is present. The inflammation primarily affects the sheath of the nerve, but often extends to the interstitial tissue, and may secondarily invade the nerve fibers. The pain is usually most severe at the sciatic notch and in the middle of the thigh.

Sciatica most commonly occurs at middle life or later, and is more frequent in males than females. It is most likely to occur in those with a tendency to a gouty or rheumatic diathesis, whatever the latter may mean. Many cases appear to follow exposure to cold and wet.

The most marked *symptom* is pain and tenderness to pressure along the nerve-trunk. The pain is, as already stated, most marked at the notch, but discrete points may be found along the course of the nerve and at its divisions into the external and internal popliteal branches. By forcibly flexing the thigh on the trunk with the leg extended, and thus stretching the nerve, tenderness at and below the sciatic notch is readily elicited. The pain may be dull and boring in character and may be absent when the patient is at rest. It may come on in paroxysms simulating a neuralgia. The onset is usually gradual. The pain may extend throughout the entire distribution of the nerve and its branches. Numbness, tingling, and formication are often complained of. Anesthesia is not found except in very severe cases. As the disease progresses, weakness and wasting of the leg muscles may develop. Trophic and vasomotor disturbances may occur, as shown by the appearance of herpes and edema.

The presence of pain along the course of the nerve, tenderness on pressure and on extension, and the absence of any other condition on *x*-ray and pelvic examinations, will be sufficient to establish the *diagnosis* of sciatica.

TREATMENT.

The treatment of sciatica may be considered under two heads: (1) The treatment of acute or recent sciatica; (2) the treatment of the chronic or established form.

The first indication in the treatment of the acute form is rest in bed. The limb should be wrapped in flannel bandages and fixed upon a well padded splint in a position of moderate extension. The intestinal tract should be emptied by a free saline purge. It is necessary that a bed-pan be used in order to maintain as perfect rest as possible, and to avoid disturbing the splint. The pain usually decreases rapidly with complete rest, and there will be little necessity for the use of morphin. If pain persists in spite of the splint, relief may be obtained by the local application of heat and by giving the coal-tar products, such as antipyrin or acetphenetidin, and, exceptionally, morphin.

Most cases are benefited by the free administration of one of the salicylates. They should be pushed to the physiologic limit, and their unpleasant effects mitigated by combining them with the bromids. Ten grains (0.65 Gm.) of sodium salicylate may be given with the same dose of sodium bromid every four hours. Later on small doses of the iodids and mercurials may be given instead of the salicylates. However, too much emphasis cannot be laid upon rest as the essential factor in the treatment of acute sciatica. The patient should continue to rest in bed for some time—several days or weeks—after the pain has disappeared.

Most patients come under treatment after the condition has become chronic, and in such instances a prolonged rest in bed for from six to eight weeks or more is necessary to obtain good results. As in the acute case, the affected limb should be put in a splint. Massage should be employed as soon as the pain subsides enough to permit it, light massage being used. A suitable form of electricity may be applied, and local heat, preferably dry heat, is of great benefit.

The patient may be given a course of salicylates followed later by the iodids, or the iodids may be given in small doses with the salicylates, from the beginning. At the same time a suitable diet, excluding red meats, sugars and starches,

should be instituted, while full feeding should be brought about by the addition of milk in gradually increasing quantities.

In intractable cases, stretching of the nerve after exposure, by surgical means, may be resorted to, although this is rarely necessary or justifiable. The same may be said of acupuncture.

COCCYGODYNIA.

Persistent pain about the coccyx is a distressing condition. It usually occurs in patients who are in an exhausted physical condition, and is often a part of a neurasthenic or hysteric syndrome. It may occur in patients with a gouty or rheumatic diathesis. In the *treatment* of this affection rest is most essential. Electricity, especially the galvanic current, is useful in many cases. If a rheumatic diathesis be determined, a course of salicylates should be given. Massage may be employed, at first gently, and later more vigorously. Resting upon an air-cushion when sitting may give relief. Some writers recommend counterirritation. However, with a thorough rest treatment, such measures are rarely necessary. Finally, we should not lose sight of the fact that disease of the bone may be present, and may require surgical interference.

VASOMOTOR AND TROPHIC DISEASES.

RAYNAUD'S DISEASE.

Raynaud's disease is characterized by paroxysmal vasomotor disturbances, most frequently affecting the digits. The first stage is one of syncope, during which the parts become cold and the seat of considerable or, perhaps, severe pain. This stage may last from a few minutes to several hours, and is followed by the gradual onset of the stage of local asphyxia, during which the part becomes congested, and varies in color from dusky blue to almost black. In this stage the pain is severe, and may become unbearable. The affected part feels cold, and sensation may be dulled or absent. After a few minutes the discoloration fades and the normal color returns gradually, the part first affected being the part first restored,

as a rule. The cyanosis changes to purple, to pink, and then to a hyperemic redness. If this stage continues for several hours, small blebs may appear upon the part affected, followed by ulceration and gangrene. The fingers and toes are the parts most frequently affected, but other regions, such as the nose, ears, lips, and patches of skin over various parts of the body may suffer. Symmetrical parts of the body are usually attacked, hence the term "symmetrical gangrene." The vasomotor symptoms vary, and may not always follow those of a typical case. The individual attack may last a few minutes, several hours, or even days. The patient may have but a single attack; the seizures may be repeated irregularly for several years. Hemoglobinuria may occur during the attack or it may be the only symptom.

No distinct cause is known. A neuropathic heredity and general debility from any cause seem to be factors. The most common exciting cause is exposure to cold. Grief, fright and trauma appear, at times, to bring on the attack. It may be associated with other vasomotor disturbances. The underlying condition is obscure. The syncope is produced by contraction of the blood vessels, the asphyxia by dilatation of the capillaries and small veins.

Everything should be done to build up the general health of the patient. Children with a neuropathic taint should be brought up to lead open-air lives and to follow outdoor occupations. Bathing should be used for its tonic effect. Coffee, tea, alcohol, and tobacco should be excluded. The diet should be liberal, and milk should be taken freely. Constipation should be guarded against. If malaria or syphilis is found in the patient, it must be treated vigorously with appropriate remedies. Quinin has seemed to be of value in cases not malarial. Sudden changes of temperature, and exposure to cold should be avoided, and warm clothing worn, with the extremities well protected. Linen socks and underwear next to the skin, with woolen socks and underwear over these, have been found of distinct advantage. Mittens are warmer than gloves for the hands.

Warm applications should be applied during the stage of syncope. Nitroglycerin is valuable during this stage. Morphin may be given if the pain is severe. In the stage of

asphyxia the part should be wrapped in cotton-wool and elevated, if possible.

Electricity may be of some benefit. This can be given in the form of the constant current, by placing the affected part in a basin of salt water, and placing the cathode in the water while the anode is applied to the spine. Cushing's plan of treatment consists of applying an elastic bandage to the limb during the stage of syncope tight enough to stop the arterial circulation; after several minutes the bandage is loosened, when the part usually becomes red. This procedure may have to be repeated in severe cases. If gangrene occur, surgical measures may be necessary.

ANGIONEUROTIC EDEMA.

This affection is characterized by circumscribed swellings of the subcutaneous or submucous tissues. The swellings may be small in extent or may affect an extremity or one-half of the face. It is known as acute circumscribed edema, giant urticaria, and Quincke's disease. It may be hereditary and is often recurrent. It is commonly associated with gastro-intestinal disturbance.

The chief *symptom* of this condition is a characteristic swelling, which usually comes on quickly without warning. It reaches its maximum in one-half to two hours. The borders may be sharply defined or shade into the surrounding tissue. The color usually is whitish or waxy. The center of the swollen area may extend one-half inch above the surrounding skin. There are few subjective sensations in the swollen region, except a feeling of fullness, stiffness, and, in some cases, burning or itching. No objective sensory changes are found. Any part of the body may be affected, although the face, extremities, and genitalia are the most common sites of the swellings. The distribution is irregular. The swellings may last a few minutes only or several hours or days. There is a pronounced tendency to recurrence. The swellings of the mucous membranes are the most troublesome, especially those of the tongue, pharynx, or larynx. The secretion of urine may be increased, and it may contain albumin and hemoglobin. Between attacks the health is usually good. In some of the congenital cases the swellings may be permanent.

The pathology of the disease is not well understood. Heidenhain believes that capillary cells play the chief rôle in lymph formation, and that morbid influences may lead to excessive secretion and production of swelling. Some think chemical processes have a causative relation. The family forms of the disease are apt to appear at an early age, and show a wider range of symptoms.

TREATMENT.

The patient should be kept in the best possible physical condition, and an effort made to remove any physical or psychic cause that tends to lessen the subject's nervous resistance. Rest, diet, exercise, and bathing all must be carefully regulated with this end in view. Many drugs have been used, but none are specific. Strychnin does good. Atropin is useful during the attack. Osler has seen good results from nitroglycerin. Cold must be avoided. With albumin or hemoglobin present in the urine, rest and a low diet should be insisted upon. Calcium chlorid or calcium bromid, 0.6 to 1 gram (9 to 15 gr.) three or four times daily have been recommended. Oppenheim speaks of two cases cured and one helped by the use of quinin. For the relief of the swellings collodion may be tried, or compression by an elastic bandage. Edema of the glottis may require scarification or tracheotomy. Spraying with a 1 : 10,000 aqueous solution of adrenalin may be tried.

INTERMITTENT CLAUDICATION.

This condition is not considered a neurosis, although some authors incline to the view that a blood-vessel spasm may be present. Others think the condition is one of arteriosclerosis. Probably a combination of the two conditions is present.

Exposure to cold, the excessive use of tobacco and alcohol, and less frequently, syphilis, are given as causes of this disease, which usually attacks men of middle or more advanced age.

The *symptoms* consist of weakness and cramps, usually of the legs, more rarely of the arms, provoked by a moderate amount of exertion. Numbness and various paresthesias are present. The severity of the attack forces the patient to rest.

In a few minutes he may be able to walk again for a short distance, the exertion, however, bringing on another attack.

In cases with sclerosis of the peripheral vessels, there is an absence of pulsation in the posterior tibial arteries or in the dorsalis pedis arteries. If the vascular sclerosis is marked in the spinal arteries the reflexes are increased, and there is bladder disturbance during the attack. The diagnosis rests upon the history of the case, the peculiar symptoms on exertion, and on examination of the dorsalis pedis and posterior tibial arteries. The course is chronic, and lasts for years. Improvement frequently takes place, but recovery is rare. The patients usually succumb to some other arteriosclerotic disturbance.

The *treatment* of intermittent claudication is unsatisfactory. The patient should have physical rest, and should abstain from alcohol and tobacco. Local vasodilators, as electric foot-baths, are recommended. Internally, the use of the iodids and the nitrites is indicated.

PROGRESSIVE FACIAL HEMIATROPHY.

This rare affection is characterized by wasting of the skin, soft parts, and even of the bones, of one side of the face. It may be limited to part of the face, or it may affect the shoulder, upper arm, and chest on the same side as the face.

The cause of the disease is not known. It usually develops between the ages of ten and twenty. Wounds of the face, extraction of teeth, and infectious diseases all have been given as causes.

The first *symptom* usually is the development of a whitish or pigmented patch about the eye, cheek, or forehead. The skin becomes thin and shiny, and often brown or yellowish. The subcutaneous tissue gradually shrinks, the affected side of the face sinks in, and the deformity becomes apparent. The hair falls out, the bones, and, to a slight degree, the muscles are affected, though RD is not present. The sebaceous glands atrophy, but their secretion is not checked, and perspiration may be normal or increased. The atrophy may affect the tongue, hand, soft palate, uvula, and gums. Neuralgic pains and paresthesias may occur in the early stages

of the disease. The skin in late stages has a roughened, wrinkled appearance. The eyeball is sunken, and the palpebral fissure narrowed. Hemihypertrophy is the opposite condition, concerning the cause of which, likewise, little is known as yet.

A degenerative neuritis of the trigeminus has been found, *e.g.,* in the case of Virchow and Mendel. Numerous theories have been advanced as to the pathology of the disease, but nothing definite is known.

The disease does not shorten life, and there is little disturbance, as a rule, beyond the deformity produced by the atrophy. No *treatment* is known to be of benefit. Tonics and galvanism may be given a trial.

SCLERODERMA.

This affection is characterized by a hardening of the skin and subcutaneous tissues, either diffuse, affecting the greater part of the body, or circumscribed in irregular patches, bands, or rings following the distribution of peripheral nerves or of spinal segments. The circumscribed form is known as morphea or Addison's keloid. The parts of the skin and subcutaneous tissues attacked undergo atrophy.

It is most common in the female sex. Age has no influence. It has been thought to follow infectious diseases, syphilis, exposure to extremes of temperature, and mental and physical exhaustion.

Nothing definite is known in the pathology of the disease. Increase of connective tissue and changes in the blood-vessels of the skin have been found. The thyroid has been found atrophied. Not infrequently a positive Wassermann is present.

The *symptoms* usually come on slowly. Before the changes in the skin the patient may complain of pains in the joints. Not infrequently, if the patient comes under early observation, a more or less marked swelling or infiltration of the skin in various parts of the body is noted. Sometimes this swelling is very marked. Later the swelling subsides, the skin begins to shrink, and a feeling of stiffness is developed in the skin, most commonly at the back of the neck, the shoulders, face,

and scalp. The disease progresses slowly, and at the height of the induration the skin of the affected area becomes leather-like in thickness. It cannot be pinched up, nor will it pit upon pressure. The underlying structures become fixed as the skin becomes rigid. The face loses all expression; the movement of the lower jaw is very limited. If the skin of the chest is affected respiration is interfered with. If the scleroderma is widespread the patient lies almost as rigid as a statue. The affected skin is not sharply separated from the sound skin; the color is usually white, but may be mottled or pigmented. The mucous membranes may be affected.

The secretions of the skin are diminished; cyanosis of the limbs is common. Deformities result late in the disease. Shortening, atrophy, and deformity of the fingers, with thickening and rigidity of the skin, give rise to the condition known as sclerodactylia. In morphea the patches resemble the diffuse form, but the rigidity is not so marked. It may be complicated by Raynaud's disease.

The outlook is best in children. Spontaneous recovery may occur in the early stages. The disease of itself is not directly fatal, but the subjects are predisposed to intercurrent affections, such as rheumatism or pneumonia.

TREATMENT.

The patient should be protected from cold and dampness. The early stages are the most favorable for the employment of salicylates, and, in given cases, of the iodids and mercurials, and, when a positive Wassermann is present, of salvarsan. Thyroid extract has been used with apparent benefit in some cases. No other glandular extracts have any noticeable effect. Galvanism has been used on the skin near the affected areas with good effect, probably by the production of hyperemia.

The patches must not be irritated for fear of ulceration or possibly further thickening. Massage and inunctions of oil are beneficial. Rubbing must be gentle, and the treatment kept up for years. Baths are of service only in so far as they promote general nutrition. Thiosinamin, either subcutaneously or by mouth, is reported of value. It can be given in capsules of 0.05 gram (gr. ¾) at a dose.

ERYTHROMELALGIA.

This affection was first described by S. Weir Mitchell in 1872. The condition is a rare one, most commonly affects men, and may occur at any age. It usually attacks the feet, and is characterized by burning pains and redness, made worse when the parts are allowed to hang. As in Raynaud's disease, the affection appears to be influenced by thermic changes. Numbers of cases are reported in association with organic nervous disease. The pathology of the disease is obscure.

The characteristic *symptoms* of erythromelalgia relate to pain in the feet, provoked by the erect posture and by locomotion. The pain may be intense, and is described as aching and burning. The affected part becomes congested and swollen and has the appearance of an active hyperemia. Profuse perspiration may occur. In the beginning the attacks are intermittent and brought on by exposure to heat, exertion, or after a pendant position. Later attacks may last for days or weeks; the parts become cyanosed, cold and often thickened, with trophic affections of the skin, nails, and even bones.

The differential diagnosis must be made from Raynaud's disease and the pain caused by flat-foot. No difficulties are as a rule presented.

The *treatment* of this condition is most unsatisfactory, the outlook as to cure being far from good. The patient should avoid overexertion and exposure to heat. Rest and elevation of the part help in relieving the pain. Stretching of the nerves supplying the parts has been done with some success. Opium may have to be relied upon for relief of pain during the attack.

ACROPARESTHESIA.

This affection is characterized by paresthesia of the distal portions of the extremities and is one of the most common vasomotor neuroses. When it attacks the hands and feet it may arise independently of all other diseases; again, it may be a partial phenomenon of various neuroses.

The condition is commonest in the female sex, usually after middle life, and in those who do scrubbing, washing, and sewing. It is prone to occur in those who are compelled to

be on their feet a great deal. The gouty or rheumatic diatheses and alcoholism appear to be factors.

The condition is supposed to be that of constriction or spasm in the peripheral arteries of the area attacked. Most cases occur in persons of a neuropathic makeup.

The symptoms include pain, numbness, tingling or coldness, usually beginning in the hands, and later affecting the arms, feet, and legs. They arise suddenly, in the morning before the patient rises from his bed. The numbness is spoken of as "waking numbness." Usually there is no muscular weakness or anesthesia. General restlessness and nervousness generally are present. Areas of local congestion and sweating are sometimes seen. The parts become pale in the early part of the attack, which may last from a few minutes to several hours, and later there is a reactive hyperemia.

TREATMENT.

The use of alcohol, tea, and coffee should be discontinued. The patient should avoid occupations in which there is irritation of the extremities by heat and cold. The diet should be plain but nutritious, and the bowels regulated. Anemia can be treated with iron and arsenic. The dietary should be appropriately regulated in gouty and rheumatic subjects, who also should undergo a course of anti-gouty therapeusis. Bromids may be of value in vasomotor irritability. Electricity in the form of galvanism, static sparks, or high frequency currents may help the pain and paresthesia. Warm salt baths and hot air baths, followed by sponging with cold water and by brisk friction, help both the local and the general condition. Various drugs, such as ergot and quinin, are thought to be of benefit. Cases showing signs of deficient thyroid function may do well on thyroid extract.

HYPERTROPHIC PULMONARY OSTEOARTHROPATHY.

This disease, described by Pierre Marie, is characterized by enlargement of the hands and feet, and of the ends of the long bones. The condition, as a rule, develops slowly, and is always associated with some chronic disease, especially of

the lungs, pleuræ, or bronchi. Some cases appear to follow syphilis, and others heart disease.

The characteristic *symptoms* of the disease are the clubbed terminal phalanges, tipped by thickened, curved, and deformed nails. Thickening of the lower end of the radius, ulna, or tibia, may be marked, and at times these structures are the seat of pain. Changes do not occur in the bones of the face or head. Effusion into the joints may occur.

The course of the disease depends upon the disease producing it; it does not of itself have a fatal termination. The treatment is that of the primary disease.

LEONTIASIS OSSEA.

Leontiasis ossea is a rare affection in which there is hyperostosis of the cranial bones. There is greatly increased thickening of the cranium and, in given instances, of the bones of the face. It occurs early in life, and Putnam regards trauma as a factor in its etiology. The pathology of the affection is not understood.

No treatment is known. Organic extracts, iodids, arsenic, and other drugs have given no result. As a rule, the process is progressive. Surgery may be able to relieve intracranial symptoms due to pressure.

OSTEITIS DEFORMANS (PAGET'S DISEASE).

This disease, prone to affect men of middle and advanced age, is distinguished by certain characteristic features, of which a large head, bowed femurs and tibiæ, and thick clavicles are the most significant.

The patient may first notice his *symptoms* by the gradual increase in the size of his head, or because of a decrease of his height. When fully developed the large skull is noticed in contrast to the narrow face. The expression is often dull, and the patients look older than their years. Ankylosis of the vertebræ or other joints is not uncommon. The hips are wide, the femurs curved outward, and the tibiæ thick and curved. The disease lasts for years. The cause is unknown. Marked arteriosclerosis is constantly present.

The *treatment* is purely symptomatic. Various organic extracts have been used with little or no effect. A case at present under observation seems to have done well for the past year on adrenalin. The treatment is practically limited to keeping the patient in the best physical condition and warding off, if possible, arteriosclerotic changes.

ADIPOSIS DOLOROSA.

This affection, described by Dercum, is a disease of adult life. It is characterized by deposits of fat in various parts of the body, the deposits being painful and tender to touch and pressure. They occur especially upon the arms, hips, chest, abdomen and back; but also upon the forearms, about the knees, and less frequently the legs. They never occur on the hands and feet, and are extremely rare upon the face; one case with deposits upon the forehead has been observed by one of the writers. Most frequently they present themselves in the form of separate masses or nodules. Less frequently, in the form of diffuse localized deposits, and least frequently in the form of a diffuse generalized adiposis. The nodules are composed of fat, and the pain and tenderness are accounted for by a localized neuritis in the fatty tissue. The nodules or masses sometimes make their appearance quite rapidly, the swelling being very painful, and for a time even indurated and hard like a "caked breast"; the induration later subsides, but the enlargement persists. Most frequently the deposits make their appearance gradually. There is muscular, and often cardiac, weakness. Spontaneous bleeding may occur from the mucous surfaces and occasionally into the fatty tissue; the latter bruises very readily. There are no sensory changes, save a slight occasional diminution of cutaneous sensibility in the affected areas. There are no characteristic changes in the reflexes. Mental changes are not noted, save in long-standing and advanced cases. Hebetude and mental slowness may then be noted, or, on the other hand, marked irritability, suspicion, confusion, and even hallucinations. A few cases have necessitated asylum commitment. On the whole, however, mental symptoms are infrequent.

In cases that are at all pronounced the prognosis is very unfavorable, the patient finally dying of cardiac weakness and general exhaustion. Autopsies have demonstrated atrophic and other changes in the thyroid gland, and, in varying degrees, in the pituitary and adrenals. Adiposis dolorosa is clearly a disease of the internal secretions.

The symptoms are quite characteristic. The only cases presenting difficulty are those in which the nodules are very small. Here a careful examination usually resolves the doubt. The affection is readily distinguished from myxedema by the fact that in the latter the head and face are early implicated, and by the absence of pain.

Early cases are greatly benefited by *treatment* with thyroid extract, and, at times, the affection is permanently arrested by this means. In severe and pronounced cases, however, thyroid administration fails, and the patient goes from bad to worse. Strict dietary measures should, of course, be instituted. The pain should be controlled by the salicylates and iodids; the cardiac and general weakness, by appropriate measures. When the pain permits, massage may be practised.

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